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10

·中英文对照·

牙周病临床评估和诊断程序

Understanding Periodontal Diseases: Assessment and Diagnostic Procedures in Practice

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内容提要

“口腔临床要点快速掌握系列”是国际著名的Quintessence出版社近期出版的介绍口腔各科基本技术和最新医学理论的专业丛书。该丛书自2002年起陆续出版发行，我社第一时间引进，以便让国内读者同步了解国际口腔技术发展的新情况。本书详细介绍了牙周病的发病机制、临床表现以及国际权威的牙周病评估标准和实用的检查方法等。本书可以帮助读者全面正确地评诂和诊断牙周疾病，进而提高牙周病的治疗效果。本书采用了中英对照的编排方式，对提高读者的专业英语水平大有裨益。本书适合于临床口腔科医师、技师和口腔医学生阅读。

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序

对牙周组织和牙周疾病的认识和理解对有效维持口腔卫生健康十分重要。本书是针对全科牙科医生Quintessentials系列丛书的第一册，其通过简洁易读的、权威且有吸引力的论述的牙医们这方面的知识。

本书中每一章内容都包括目的、要点、对正文进行高质量补充的图解说明以及对扩展阅读的建议，这些都是精萃系列丛书吸引人的特点。本书涵盖了丰富的牙周病学信息和治疗指导，这是临床上高质量地治疗牙周病必不可少的。本书适合所有的口腔工作者和口腔医学生。

随着口腔保健意识的提高，患者一生能保留越来越多的牙齿以及这些牙齿拥有更长的寿命，本书中的牙周病学内容是成功临床治疗的重要因素，对那些努力提高患者口腔健康的牙医，本书及Quintessentials系列丛书提供了最有价值的财富——应用最新知识和认识以及牙科学新进展为患者服务的方法。我向您推荐这本优秀的图书。

主 编 **Nairn H F Wilson**

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前　言

本书是致力于为全科口腔医师提供治疗牙龈炎和牙周炎全面而实用的治疗指导的五本书中的第一本，本书名为《牙周病临床评估和诊断程序》，它使读者在牙周炎的评估和诊断过程中运用逻辑思维，以避免诊断失误和分辨牙周炎的危险人群。它还对相关的解剖结构和疾病过程中健康和病理状态的临床表征，以及疾病的分类等作出全面的修订总结，从而使读者通过临床诊断原则，包括放射检查和其他特殊检查，达到对牙周疾病诊断的目的。

希望读者在读完本书后能够有以下收获。

- 理解最新的牙周疾病进展、分类和临床过程。
- 理解牙周病易感性评估的概念和易感因素，并能指出由于牙周炎导致失牙患者的易感水平。
- 识别健康和病理牙周组织（在广义的范畴），并能将其按特征分类。
- 诊断普通的牙龈疾病和牙周疾病。
- 通过简单的牙周诊断检查方法，包括放射检查和其他特殊检查，来对疾病进行诊断。
- 通过相应的放射检查对主要的病理变化作出简明的报告。
- 理解特殊牙周病诊断需要的特殊检查及其检查结果的意义。
- 对影响牙周组织的疾病进行广义的分类，并对其进行鉴别诊断。
- 了解牙周疾病的诊断在今后十年可能的发展方向。

Iain L C Chapple
Angela D Gilbert

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第 1 章 牙周膜

A Whistle–Stop Tour of the Periodontium

目 的

这一章的目的是复习牙周膜的重要解剖学和组织学结构特征。

Aim

This chapter aims to provide the practitioner with a contemporary review of the important anatomical and micro-anatomical features of the periodontium.

要 点

通过本章的复习，实践者应能识别在牙周组织检查中需要评估的关键的临床特征，因为它们能帮助进行诊断和制定治疗计划。

Outcome

At the end of this chapter the practitioner should be able to identify key clinical features that need to be assessed during the examination of patients' periodontal tissues, since these features help inform diagnostic and treatmentplanning processes.

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Terminology and Orientation

The periodontal tissues form the supporting apparatus of the teeth. Their role is to protect the teeth from masticatory forces and infection, thereby facilitating normal oral function and preventing premature tooth loss. As modern medicine and standards of health have prolonged human life expectancy the periodontal tissues have to perform these functions over considerably

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牙周病与心脏、脑血管疾病之间的关系）以及与更长时间的保留牙齿有关的慢性细菌性刺激才刚开始被人们认识。所以牙龈退缩、牙齿过敏和牙齿松动成为牙医和患者每天面对的问题。健康的牙周组织如图1-1所示，其组成包括：

more years than they were designed for, and therefore recession,sensitivity and tooth mobility are daily management problems for the dental practitioner and patient. Additionally, the nature and extent of systemic problems that are created by a compromised periodontal attachment (e.g.the established link between periodontal disease and cardio-/cerebro-vascular disease) and the chronic microbial stimulus associated with retaining teeth for longer, is only just being realised. The healthy periodontal tissues are identified in Fig 1-1 and comprise:

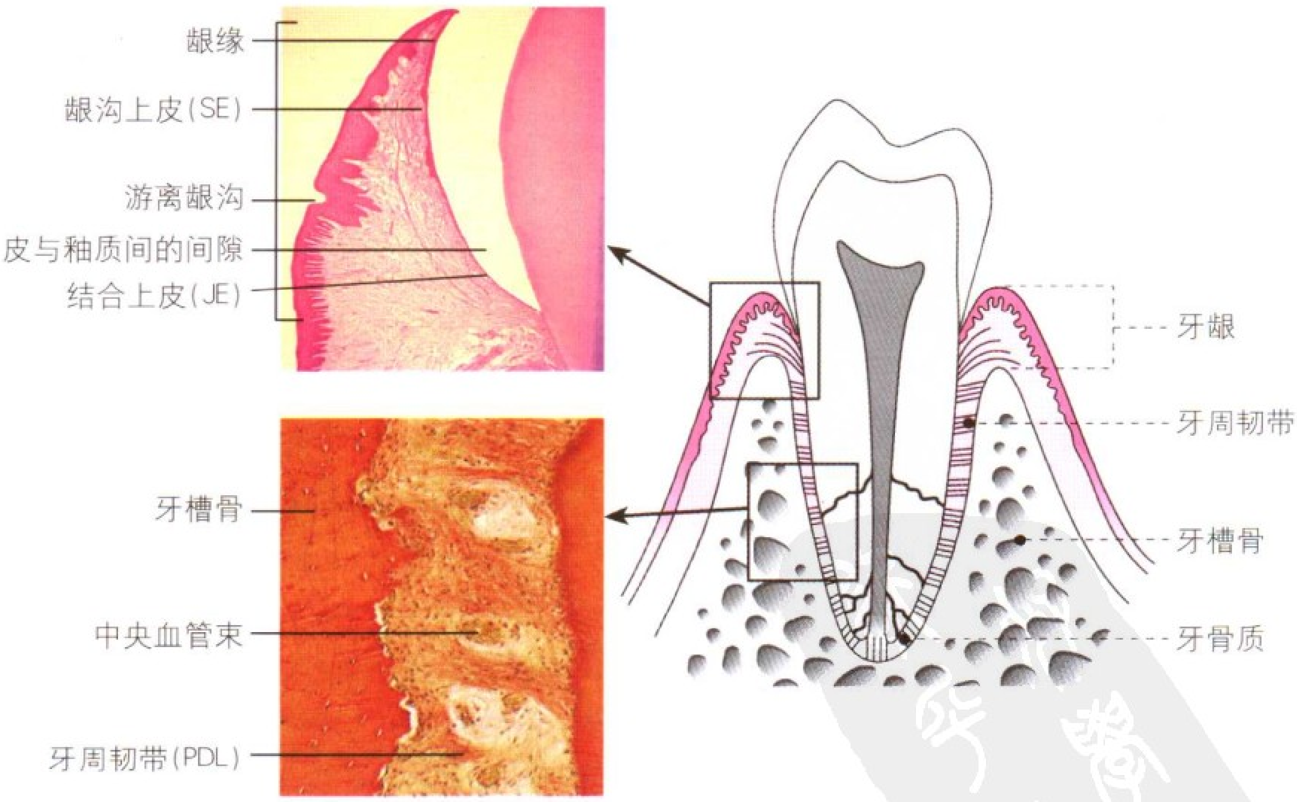


图 1-1　前磨牙牙周组织纵剖面及组织学应用解剖

Fig 1-1　Schematic longitudinal section of a premolar and associated periodontal tissues. Applied anatomy is demonstrated alongside histology (photomicrographs) of key areas.

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- 牙龈
- 牙槽骨
- 牙周韧带
- 牙骨质

牙　龈

牙龈包括：

- 龈缘——可见的牙龈边界
- 龈沟——正常龈沟深度为0.5～3 mm
- 游离龈——覆盖于牙槽嵴顶的可动的领圈状或袖口状牙龈
- 附着龈——1～9 mm 长，由牙龈-牙复合体的胶原纤维附着于牙槽骨和牙骨质

游离龈向附着龈移行的界限常可见到，在原始状态的健康牙龈上称为龈沟（图1-1和图1-2）。然而由于大多数正常口腔都可探查到一定程度的龈缘菌斑和邻面菌斑，游离龈在组织学上都有一定程度的炎症，所以临床健康和原始状态的健康被认为是不同的状态。在临床健康状态，可能存在非常轻微的炎症，所以不是总能看清龈沟。

- investing gingival complex (gingivae)
- alveolar bone
- periodontal ligament
- root cementum.

The Gingivae

The gingivae comprise:

- a gingival margin – the visible edge of the gingiva
- a gingival sulcus – (or crevice) which in health is between 0.5 and 3mm in depth
- the free gingiva—a mobile cuff of gingiva lying above the alveolar crest
- the attached gingiva—a band of 1-9mm in length, which is bound down to the underlying alveolus and cementum, by collagen fibres of the dentogingival complex.

The line at which the *free gingiva* becomes *attached gingiva* is usually visible, under conditions of *pristine* gingival health, as the *gingivalgroove* (Figs 1-1 and 1-2). However, since the majority of healthy mouths have detectable levels of marginal and interproximal plaque present, they also have a degree of inflammation present histologically, hence *clinical health* and *pristine health* are now recognised as different conditions. In clinical gingival health, it is accepted that there may be very mild inflammation present and the gingival groove is not therefore always discer-

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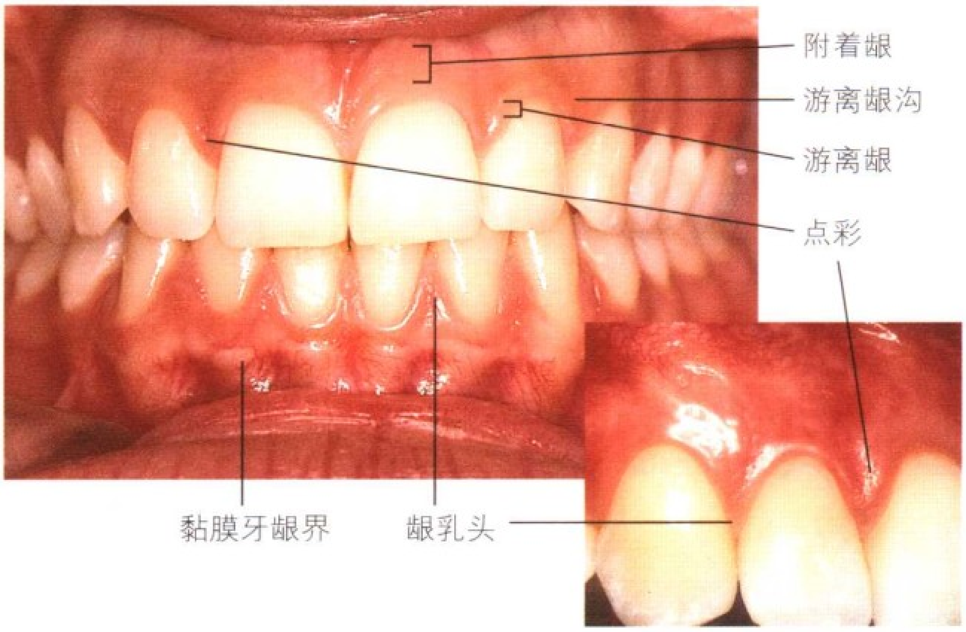


图 1-2　健康牙龈应用解剖特征

Fig 1-2 An anterior view of "pristine gingivae" demonstrating applied anatomical features from Fig 1-1.

牙龈组织是正角化上皮，在健康状态比非角化的口腔衬里黏膜更苍白或粉红。牙龈与口腔黏膜在黏膜牙龈界（MGJ）处相连，由于腭侧黏膜是完全角化上皮，所以在腭侧看不见膜龈联合。过去常常认为当龈缘由口腔黏膜构成（如牙龈退缩时），而不是由角化的牙龈构成时，牙龈边缘对刷牙时的创伤性刺激具有较差的抵抗力，然而，研究表明，在菌斑控制较好时，非角化的口腔黏膜形成的龈缘与角化的牙龈上皮形成的龈缘同样能保持稳定。

nible.

The gingival tissues are orthokeratinised and therefore, in health, appear paler or pinker than the lining oral mucosa, which is non-keratinised. The gingiva joins the oral mucosa at the *mucogingival junction* (MGJ), which is not visible in the palate, since palatal mucosa is entirely keratinised. It used to be thought that when the gingival margin was formed by oral mucosa (e.g.due to recession) rather than keratinised gingiva, the marginal tissues were less robust and resistant to the trauma of toothbrushing. However, studies have shown that a gingival margin formed by non-keratinised oral mucosa is as capable of retaining stability as a margin formed by

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游离龈和牙体组织在健康状态下被龈沟分隔，龈沟深度为0.5～3 mm（推荐的探诊力量为20～25 g或0.2～0.25 N）（图1-3）。如果探查健康的龈沟时探诊力量过大，探针将穿过龈沟底，进入结缔组织。健康状态下龈沟内分泌龈沟液（GCF），龈沟液以0.2 μl/h的速度分泌，每天分泌约1 ml并成为唾液的一部分。正常龈沟液是一种血清渗出液，是由牙龈内的动脉毛细血管内的血清被动渗出形成（图1-4），通过牙龈结缔组织进入龈沟（图1-5）。正常情况下，GCF包含血清含有的所有成分（除红细胞外），另外，龈沟液内还可有活性中性多形核白细胞（PMNLs）。炎症感染时，龈沟渗出液更像炎症渗

keratinised gingiva, provided plaque control is good.

The free gingival tissues are separated from the crown of the tooth in health by the *gingival sulcus* or crevice, which on clinical probing varies from 0.5 to 3mm, for recommended probing pressures (20-25g or 0.2-0.25N) (Fig 1-3). If a healthy crevice is probed too firmly, the probe penetrates the base and enters the connective tissues. The crevice is washed out in health by *gingival crevicular fluid* (GCF) which flows out at a rate of 0.2μl per hour contributing lml per day to saliva. GCF is a serum *transudate* in health and is formed by serum moving passively from the arteriolar capillaries of the gingiva (Fig 1-4), through the gingival connective tissues and

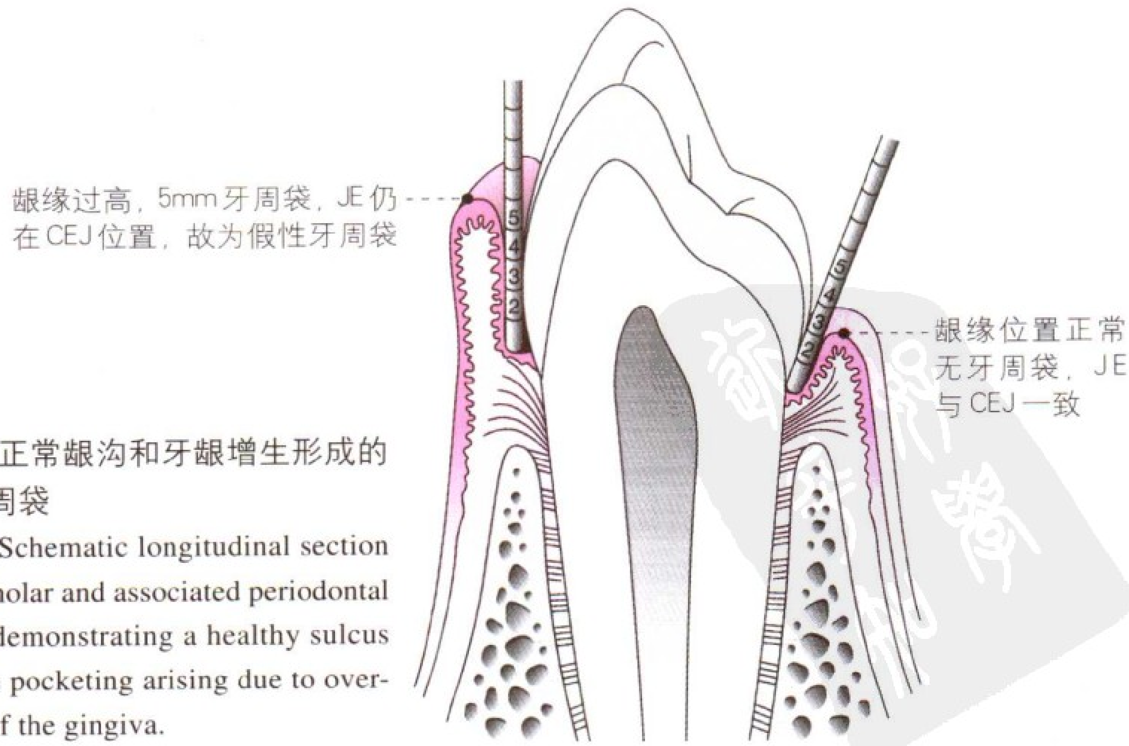


图1-3 正常龈沟和牙龈增生形成的假性牙周袋

Fig 1-3 Schematic longitudinal section of a premolar and associated periodontal tissues, demonstrating a healthy sulcus and false pocketing arising due to overgrowth of the gingiva.

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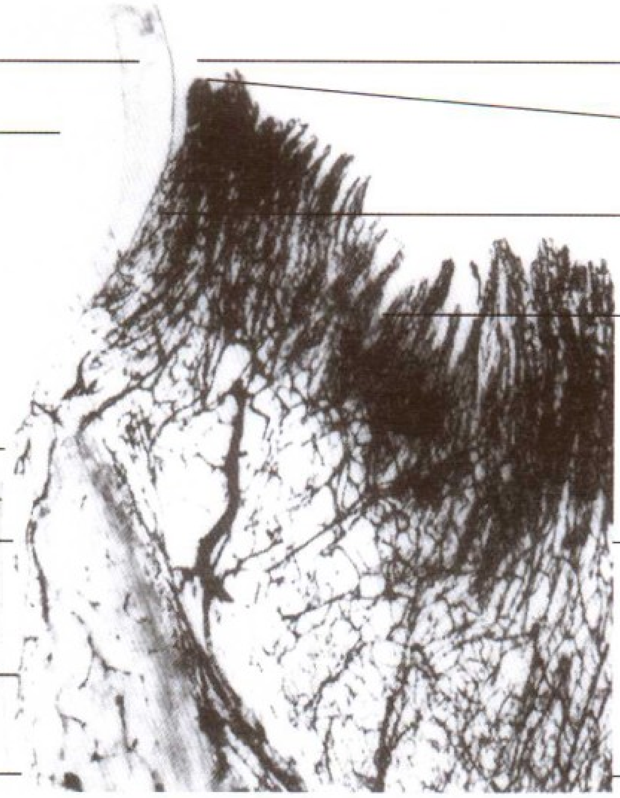


图 1-4 狗牙周膜丰富的牙龈血管染色黑白显微照相。JE 和 SE 下的毛细血管网称为龈牙丛，血清从此进入毛细血管后的静脉或进入龈沟形成龈沟液

Fig 1-4 A black-and-white photomicrograph from the periodontium of a dog following injection of dye to demonstrate the rich gingival vasculature. Large vessels (arterioles) branch off a vast network of capillaries which formloops beneath the connective tissue rete ridges of the oral epithelium. The capillary network beneath the JE and SE is called the dentogingival plexus and serum from this plexus either returns to the post-capillary venules, or it enters the gingival crevice as GCF.

出液，局部的炎症成分进入龈沟液并增加龈沟液的流速和量。

into the gingival crevice (Fig 1-5). In health, GCF contains everything serum contains, except for red blood cells, and, in addition, viable neutrophils (polymorphonuclear leucocytes; PMNLs) can be collected from it. During inflammation, the transudate becomes more like an inflammatory *exudate* and local components of that inflammatory process enter the GCF which increases in flow rate and volume.

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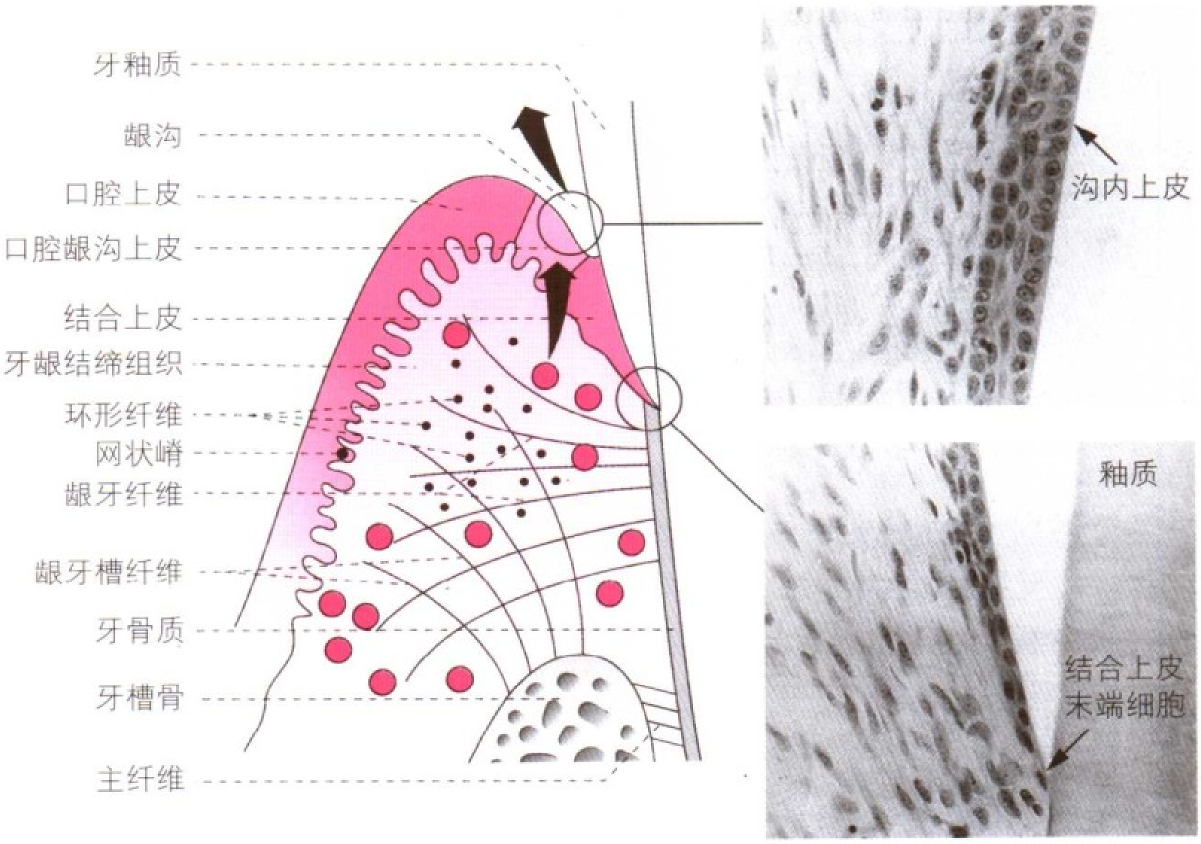


图 1-5 牙龈胶原纤维和 SE 及 JE 的正常组织显微照片。细胞间的距离较宽并逐渐变薄，在结合上皮顶端为单个结合上皮终末细胞

Fig 1-5 A schematic view of the gingivae demonstrating the gingival collagen fibre complexes. The SE and JE are also represented alongside in two photomicrographs demonstrating normal histology. Note how widely spaced the cells are and how they thin out forming a single "terminal cell" of the apex of the JE.

龈沟内衬以高度特异性的上皮组织，称为龈沟上皮（SE）和结合上皮（JE）（图1-5）。正常情况下它们紧紧地附着于牙冠，所以原始健康状态下没有龈沟除非将探针从龈缘置入。结合上皮是一种独特的结构，通过半桥粒附着于骨的内在的部分（被覆有牙槽骨的牙齿）。胚胎学上牙釉质来源于外胚层（内釉上皮），该组织是一种平常的结构。但是一旦出现附着丧失（不管是牙龈退缩还是真性牙周袋形成），结

The gingival crevice is lined by highly specialised epithelia called sulcular epithelium (SE) and junctional epithelium (JE) (Fig 1-5). These normally adhere tightly-to the crown, such that in *pristine* health no sulcus exists unless a probe is placed down from the gingival margin. The JE is unique as it forms an epithelial attachment (via hemi-desmosomes) to an internalised part of the skeleton (tooth with its investing alveolar bone). As enamel is derived

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合上皮会在来源于间充质的牙骨质上迁移导致牙骨质暴露，结果类似于开放性骨折，骨从上皮内冒出，使人体内部结构暴露于外部环境。结合上皮对于携带有宿主防御细胞（主要是多形核白细胞）和其他各种炎症成分和免疫反应成分如补体和抗体的龈沟液具有渗透性。结合上皮细胞间具有较大间隔以利于龈沟液的渗透，相反这种结构使细菌产物也能进入牙龈组织并刺激炎症反应的发生。健康状态下，该反应被控制，不会出现肉眼可见的炎症反应，但当菌斑在龈下聚集以后，炎症在结合上皮下的牙龈结缔组织内逐步进展最终形成微溃疡，当进行牙周探查时，血液通过微溃疡从牙龈结缔组织内流出进入龈沟，形成探诊出血(BOP)这一重要的临床表征。

from ectoderm embryologically (inner enamel epithelium) such a union is not unusual. However, once attachment loss has occurred (either recession or true pocket formation), the JE migrates onto cementum which is derived from mesenchyme. This results in a situation rather like a compound fracture of bone, where bone emerges from epithelium exposing the internal structures of the body to a hostile external environment. For this reason, the JE is permeable to GCF carrying the host's defence cells (mainly PMNLs) and various other components of the inflammatory/immune response, such as *complement* and *antibody*. The JE cells are widely spaced to facilitate this (Fig 1-5), but the downside of this arrangement is that bacterial products can also pass back into the gingival tissues and stimulate an inflammatory reaction. In health, this reaction is controlled and visible inflammation does not occur. However, as plaque accumulates subgingivally, the inflammation progresses within the gingival connective tissue underlying the JE and the latter eventually develops microscopic ulcers. When probed, blood can pass from the gingival connective tissues via the micro-ulcerations and can enter the crevice, creating the important clinical *sign of bleeding on probing* (BOP).

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牙龈上皮

牙龈上皮包括：

- 口腔上皮
- 口腔龈沟上皮
- 结合上皮

口腔上皮（图1-6）是一种复层鳞状上皮，起始于柱状的基底细胞，这些幼芽细胞分裂形成向上进入中心区域的细胞（棘层和颗粒层内的五角/六角形细胞），最终在表层变得扁平并失去细胞核，表层细胞积聚形成称为角蛋白的一种非渗透性蛋白，坏死后沿牙龈表面形成一种无细胞核非渗透性的角化层（正角化）。有超过20种不同类型的角蛋白，这种上皮细胞的角蛋白以它们的来源为特征（例如牙龈上皮表达细胞角蛋白—K1，2，5，6，10，12，16）。基底细胞层在结缔组织脊（称

The Gingival Epithelium

The gingival epithelium comprises:

- oral epithelium
- oral sulcular/crevicular epithelium
- junctional epithelium.

The oral epithelium (Fig 1-6) is a stratified squamous epithelium with several layers, starting with the columnar basal cells. These germinal cells divide to produce cells that move up to the central zone (pentagonal/hexagonal cells within the *stratum spinosum* and *stratum granulosum*), which ultimately flatten at the surface and lose their nuclei. The surface cells accumulate an impermeable protein called keratin and upon death they lie along the gingival surface forming a non-nucleated and impermeable keratinised layer (ortho-

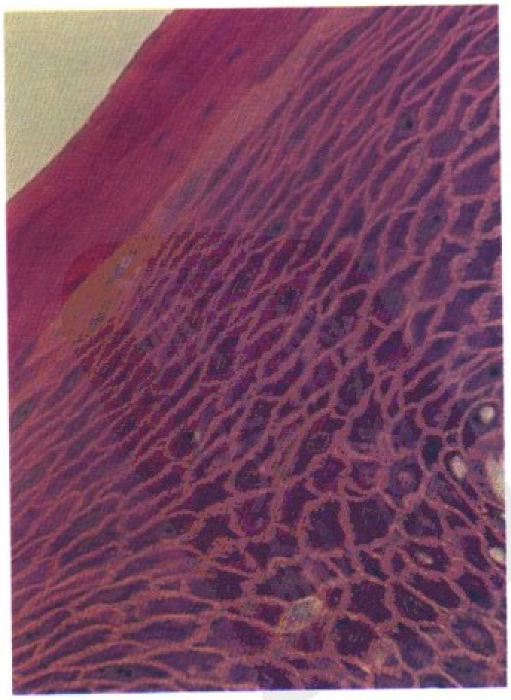


图1-6 牙龈上皮显微照片：柱状基底细胞和棘层星形细胞以及扁平表层角化细胞。注意表层细胞无细胞核且完全角化形成阻止微生物入侵的渗透屏障

（引自Riviere. *Lab Manual of Normal Oral Histology*. Quintessence：Chicago，2000）

Fig 1-6 Photomicrograph of gingival epithelium from the facial surface demonstrating columnar basal cells (just in view) and pentagonal cells of the stratum spinosum, with flattened surface cells which have accumulated keratin. Note the absence of nuclei at the very surface, which is pure keratin and forms the permeability barrier to prevent microbial invasion.

(From Riviere. *Lab Manual of Normal Oral Histology*. Quintessence: Chicago, 2000)

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网状脊或丛状脊）的移行遵循类似"滑行轨道"的过程，网状嵴用以增大上皮与结缔组织的接触面积。口腔牙龈上皮通过形成对细菌及其产物非渗透性的物理屏障，成为固有的免疫反应的重要部分，且浸润于含有各种酶（如溶菌酶）和免疫球蛋白的唾液中，当口腔上皮（在龈缘内）向下移动，就形成龈沟上皮。

龈沟上皮是口腔上皮与结合上皮的连接部分，其基底细胞层没有网状嵴，基底层迅速分裂，加速表层的流动置换，排出附着于龈沟上皮细胞内的细菌，所以这些细胞没有足够时间积聚角蛋白，且表层细胞角化不全（它们仍然具有细胞核），最后当它们接触牙齿时会失去角蛋白，由于此点它们成为高度特异化的结合上皮且表达K19和K13，龈沟上皮细胞和结合上皮细胞均为"活跃的上皮细胞"，对细菌及重要的细胞信号分子产物——细胞因子（如IL-1）具有胞饮作用（图1-7），细胞因子是化学信息传递者，它能在牙龈结缔组织内发动早期炎症变化。

keratinisation). There are over 20 different types of keratin and the keratin profile of epithelial cells characterises where they come from (e.g. gingival epithelium expresses cytokeratun- Kl, 2,5,6,10,12,16). The basal layer of cells follows a "roller-coaster" course as they travel over connective tissue ridges called "rete ridges" designed to increase the surface area of epithelial-connective tissue contact. The oral gingival epithelium forms a key part of the innate immune response by forming an impermeable physical barrier to bacteria and their products, which is bathed by saliva containing a variety of enzymes (e.g. lysozyme) and imunoglobulins (antibody). As the oral epithelium drops down inside the gingival margin, it becomes SE.

The SE is midway between the oral epithelium and the JE. There are no rete ridges for the basal cells to conform to (Fig 1-5). The basal layer divides rapidly, increasing turnover of the surface layer to help shed bacteria that attach to epithelial cells within the gingival sulcus. As a result these cells have less time to accumulate keratin and surface cells are para-keratinised (they still have nuclei), eventually losing their keratin surface as they touch the tooth. At this point they become highly specialised JE and express K19 and K13 in addition to the more ubiquitous keratins. Both the sulcular epithelial cells and junctional epithelial

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cells are "active epithelial cells", capable of pinocytosis of bacteria (Fig 1-7) and production of important cell signalling molecules called cytokines (e.g. interleukin 1– IL-1), which are chemical messengers that set up early inflammatory changes in the underlying gingival connective tissues.

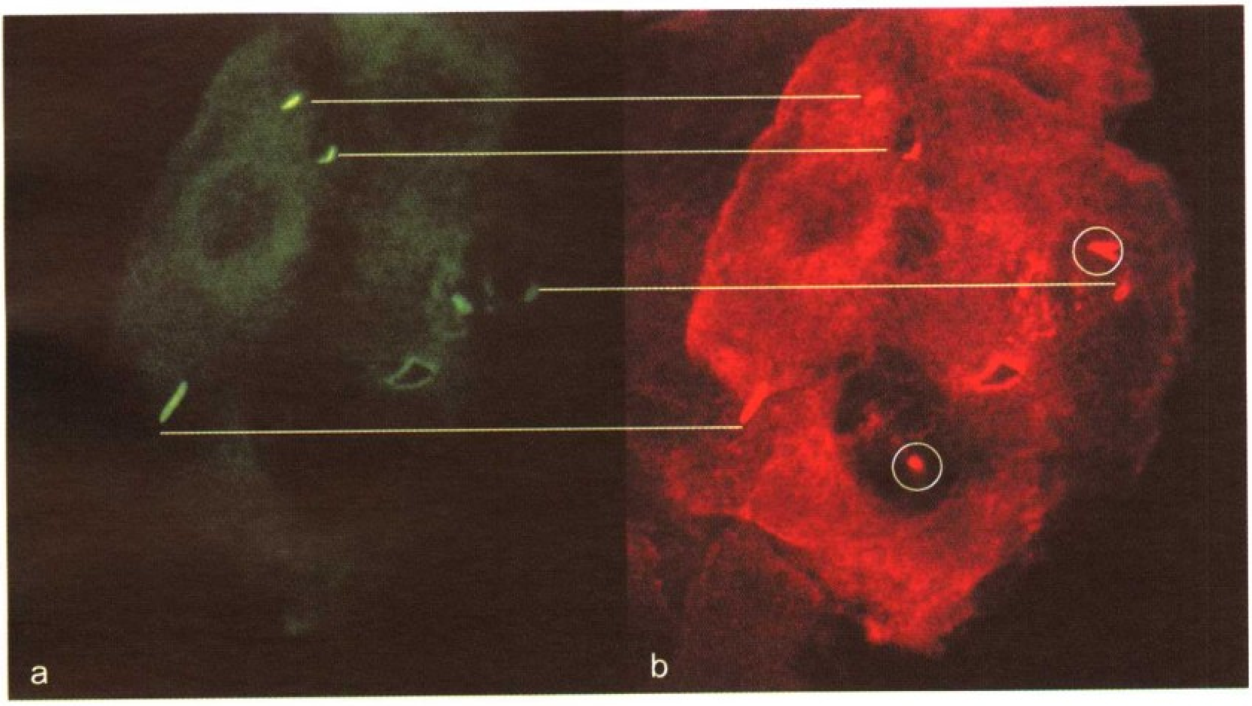


图 1-7 a. 感染福赛类杆菌的龈沟上皮。绿色荧光标记的是抗福赛类杆菌抗体，在有绿色滤光器的显微镜下，绿色荧光染料标记细胞外的福赛类杆菌；b.红色若丹明标记抗福赛类杆菌抗体，上皮细胞打孔后抗体进入。红色染料对细胞外和细胞内的福赛类杆菌均能着色，所以细胞外的细菌可有红色和绿色两种标记，细胞内的细菌只能用红色标记。注意在经胞饮作用后上皮细胞内的细菌可在静脉内出现

Fig 1-7 (a) A single sulcular epithelial cell curetted from a patient who harboured the pathogen *B. forsythus*. Green fluorescein-labelled secondary antibody raised against *B.forsythus*. Under a microscope with a green filter, the green fluorescein dye labels *B.forsythus* located outside the cell.
(b) Red rhodamine-labelled secondary antibody raised against *B.forsythus* after holes were punched into epithelial cell membrane to allow antibody inside. The red dye stains *B.forsythus* located both inside and outside the cell. Thus, bacteria located outside the cell take up a double label and stain both red and green. Those bacteria located inside the cell stain with the red label only. Note the bacteria inside the epithelial cell appear inside vacuoles after pinocytosis.

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结合上皮是一种高度特异性的上皮组织，细胞是非角化的细胞，比其他任何普通上皮分化速度都快（口腔上皮1个月更新一次，而结合上皮2～6 d更新一次），结合上皮的变化是决定牙龈炎向牙周炎转化的关键。健康状态下结合上皮附着于牙釉质，纵剖面观，从20～30个细胞厚度向结合上皮根方逐步递减，直至根方于釉牙骨质界处只有1个细胞厚度。在牙周炎早期，当牙周附着（韧带和牙骨质）受到损坏时，结合上皮向牙齿根方移行，来分隔生活的内在组织和龈沟内潜在的有害细菌及其产物。结合上皮的根向移行是牙周附着丧失的首要临床标志，其结果是真性牙周袋的形成。当龈缘冠向膨胀（如药物性牙龈增生见图1-3）时形成假性牙周袋，龈沟深度大于3 mm，但结合上皮的根方细胞仍保持在釉牙骨质界处，并没有附着丧失。计算临床附着水平可用下面一个简单公式：

临床附着水平（CAL）＝牙周袋探诊深度（PPD）＋牙龈退缩量

The JE is a highly specialised epithelial tissue whose cells are non-keratinised and divide faster than any other normal epithelium (2-6-day turnover, compared to a month for oral epithelium). The JE is the key to determining when *gingivitis* becomes *periodontitis*. It is positioned onto enamel in health and, in longitudinal section, tapers from 20 to 30 cells thick to the apical cell of the JE (Fig 1-5), which is a single cell positioned at the CEJ. As the underlying periodontal attachment (ligament and cementum) becomes damaged during early periodontitis, the JE migrates down the root surface in an effort to *wall off* the vital internalised tissues from the potentially hostile bacteria within the crevice and their products. The *apical migration of the JE* is the first clinical indicator of *periodontal attachment loss*, and results in the formation of a *true pocket*. A *false pocket* arises when the gingival margin expands coronally (e.g. with drug-induced gingival overgrowth–Fig 1-3), the crevice deepens beyond 3mm, but the apical cell of the JE remains at the CEJ and there has been no *attachment loss*. A simple formula is used to define the *clinical attachment level* (CAL):

CAL = probing pocket depth + recession

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牙龈退缩量测量的是釉质牙骨质界（CEJ）到龈缘的距离。假如龈缘在CEJ的根方则称为牙龈退缩，如果是在CEJ冠方则为牙龈增生和假性袋形成（图1-3），显然我们假设健康状态时龈缘位于釉质牙骨质界，而实际上龈缘位于CEJ冠方并通过半桥粒体附着于CEJ上2～3 mm（图1-1）。真正的退缩应为从CEJ到根面上皮附着点的距离（图1-8），但是在临床上是看不见的，故用临床附着水平来代替组织学附着水平以弥补此点。临床退缩是指龈缘到CEJ间的可视间隔，临床牙周袋的测量是牙周袋探查检测，称为牙周袋探诊深度（PPD）并非真正的牙周袋深度。另外在临床上我们还能遇到一些类似的情况，如“正常的”龈缘位置、临床解释时用到的增生肥大等。

where recession is the distance measured from the CEJ to the gingival margin. If the gingival margin is apical to the CEJ then this is recession; if it is coronal to the CEJ then this is "overgrowth" and false pocketing (Fig 1-3). Clearly, this assumes that the gingival margin is at the CEJ in health, when in fact it lies coronal to the CEJ and is broadly attached by hemidesmosomes up to 2–3mm above the CEJ (Fig 1-1). True recession should be measured from the CEJ to the point of epithelial attachment to the root surface (Fig 1-8), but this is not visible clinically and hence the term "*clinical attachment level*" rather than "*histological attachment level*" is used to overcome this. Clinical recession is a visual gap between the gingival margin and CEJ and clinical

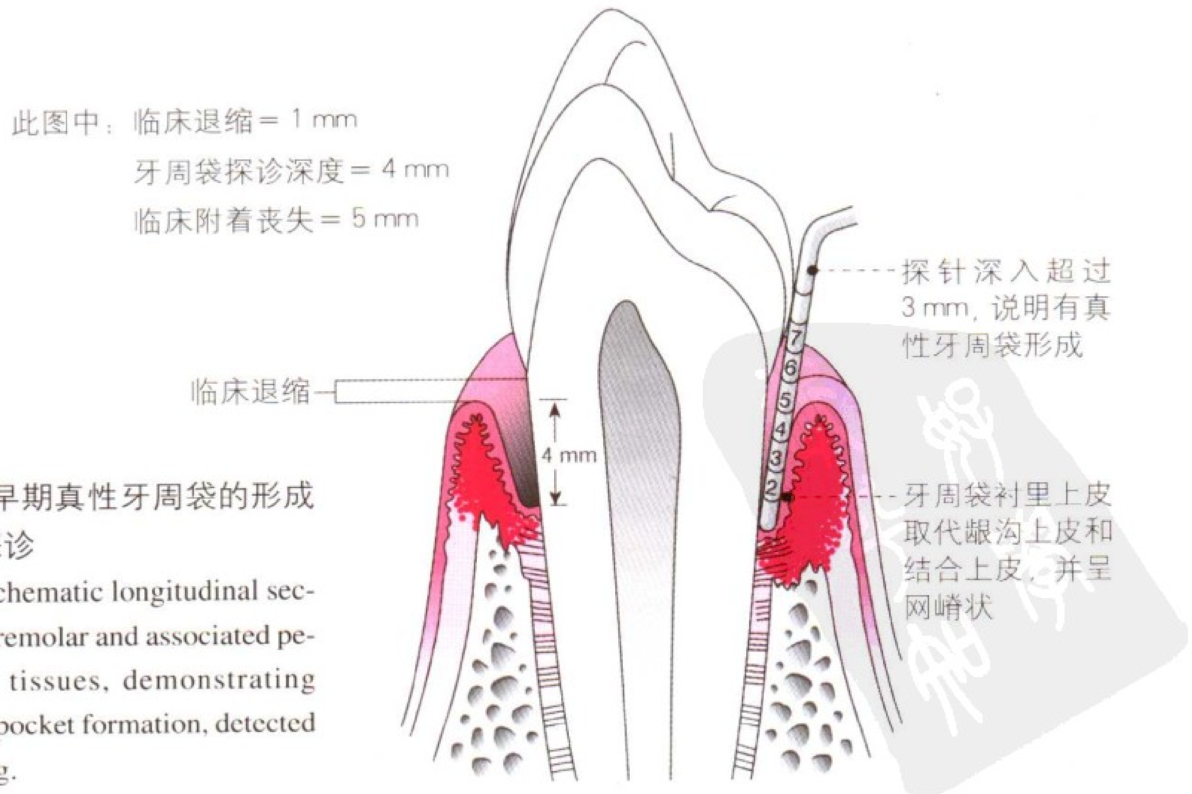


图1-8 早期真性牙周袋的形成及牙周探诊

Fig 1-8 Schematic longitudinal section of a premolar and associated periodontal tissues, demonstrating early true pocket formation, detected by probing.

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pocketing is measured by probing (hence the term probing pocket depth (PPD), rather than *true* pocket depth). It can also be seen that what represents the "normal" position of the gingival margin and what represents "overgrowth" are open to clinical interpretation.

邻间龈乳头覆盖于邻间牙槽嵴，这是一个脆弱的结构部分，因为其血供的解剖结构，该处颊、舌侧的微血管在狭窄的峡部相连。健康状态龈乳头的形状决定于牙的接触关系，当接触紧密时，龈乳头呈正常的三角形（三维状态呈锥体状）（图1-2），然而，如果牙齿被分开，龈乳头将失去其三角形（图1-9），但组织仍可呈健康状态。颊舌侧龈乳头在牙邻间相遇的区域，称龈谷，位于牙齿触点下。该区域由于上皮为非角化且口腔卫生措施难以达到（很少人常规清洁邻面）。因此该区域成为最薄弱的地方，是疾病最易发生和进展的区域。因此牙龈炎症易于在该区域首先发生，继而出现邻间骨丧失。

The interdental papilla covers the interdental bone crest and is a fragile structure due to the anatomy of its blood supply, where buccal and lingual micro-vessels meet in a narrow isthmus. In health the papilla shape is determined by the tooth contacts, and it is normally triangular (pyramidal in 3-dimensions) due to tight tooth contacts (Fig 1-2). However, if the teeth are spaced apart, the triangularity is lost (Fig 1-9), but the tissue may still be healthy. Where the facial and lingual papillae meet within the interdental zone, is a "col", situated beneath the contact point of the tooth. This area is the most vulnerable to disease initiation and progression, as the epithelium is non-keratinised and the col-area difficult to access for oral hygiene (very few people routinely clean interproximally). As a result of this, gingival inflammation tends to initiate in this area and interdental bone loss follows.

牙龈结缔组织

牙龈结缔组织基本包括基质，血管、淋巴管和神经组织，为一系列分别

The Gingival Connective Tissues

The gingival connective tissues essentially comprise ground substance, blood and

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图 1-9　健康牙龈邻间龈乳头形状。如图所示前牙窄的触点和龈谷区、宽的磨牙触点和深且脆弱的磨牙邻间龈谷区

Fig 1-9 Clinical photograph of healthy gingivae, demonstrating the effect of the tooth contact area in dictating interdental papilla shape. The schematic diagrams show the narrow contact point and narrow col between incisors and the broader contact and deeper, more vulnerable col area between molar teeth.

被称为牙龈纤维的胶原纤维束横切，后者包括：

- 龈牙纤维（图 1-5）
- 龈牙槽纤维（或牙槽嵴纤维）（图 1-5）
- 环形纤维（环或绕牙龈纤维）（图 1-5）
- 越隔纤维（或邻间纤维）（图 1-10）

lymph vessels and neural tissue, transsected by a series of collagenous fibre bundles called the *gingival fibres*. The latter comprise:

- dentogingival fibres (Fig 1-5)
- alveolar-gingival fibres (or alveolar crest fibres) – (Fig 1-5)
- circumferential fibres (circular or circum-gingival fibres) – (Fig 1-5)
- transseptal fibres (or interdental fibres) – (Fig 1-10).

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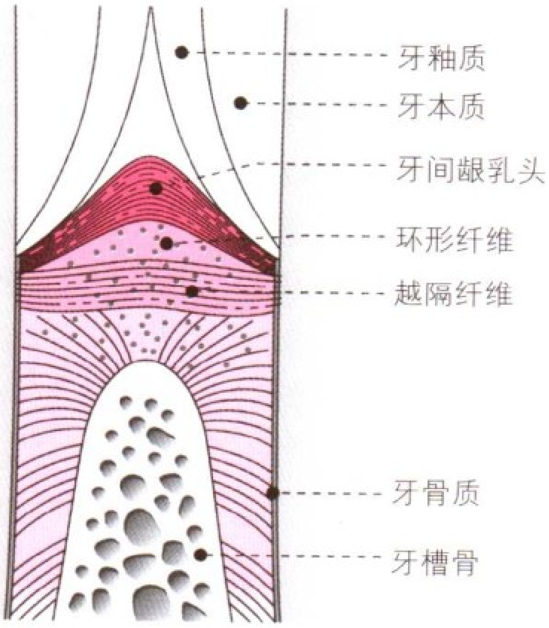


图 1-10 越隔纤维和环行纤维截面

Fig 1-10 Schematic facial view of the interdental area showing the transseptal fibres and cross-section of the circular fibres.

主要的细胞是成纤维细胞，它产生胶原和胶原酶来代替胶原，甚至在健康状态下代替一些炎症细胞（多形核白细胞，单核细胞，T、B淋巴细胞）。牙龈纤维的作用是使牙龈紧贴牙齿形成牙龈袖口，限制龈下菌群的移植，当牙龈出现炎症时，它们被炎症渗出充满（水肿），血管膨胀，纤维被拉长并导致牙龈松弛，变得圆钝和肿胀（见第5章）。

The main cells present are fibroblasts which produce collagen and the collagenases to replace collagen, and, even in health, some inflammatory cells (PMNLs, monocytes, T- and B-lymphocytes). The purpose of the gingival fibres is to maintain a tight gingival cuff and close adaptation of gingiva to tooth, restricting subgingival microbial colonisation. When the gingivae become inflamed, they fill with inflammatory exudate (oedema), blood vessels dilate, and the fibres become stretched resulting in the gingivae loosening, and becoming rounded and swollen (see Chapter 5).

牙周附着装置

牙周附着装置包括：

The Periodontal Attachment Apparatus

The periodontal attachment apparatus describes:

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- 牙骨质——正常情况下止于CEJ，但也可延伸到牙釉质
- 牙周韧带（PDL）——正常宽度为0.2 mm
- 牙槽骨——上颌骨或下颌骨的一部分，并形成牙槽窝

- root cementum – which normally finishes at the CEJ but can extend onto enamel
- periodontal ligament (PDL) – which is 0.2mm wide in health
- alveolar bone – that part of the mandible or maxilla that forms the tooth socket.

牙骨质

牙骨质是一种骨样组织（含有约50%矿物质，不含血管、淋巴和神经组织），覆盖牙根保护根面牙本质，防止牙周成纤维细胞分泌的胶原酶的破坏，使重要的牙周韧带纤维固定在牙根表面。牙骨质冠方较薄（距CEJ 0.05～0.1 mm），向根尖方向渐厚（0.2～1 mm）。牙骨质有两种类型：

- 无细胞性牙骨质——在牙齿发育过程中紧贴牙根牙本质形成，并围绕插入的牙周韧带纤维—穿通纤维（图1-11）。
- 细胞性牙骨质——在陷窝（腔隙）中有牙骨质细胞并在功能行使过程中在无细胞性牙骨质的表面形成。

理论上牙骨质应终止于CEJ（图1-12），但也可短或不足（图1-13）（牙

Root cementum

The cementum is a bone-like tissue (approximately 50% mineral, but containing no blood, lymph or neural structures) that covers the root and protects the root dentine from collagenases released by periodontal fibroblasts. But importantly it anchors the ends of the PDL fibres to the root. It is thinner at the coronal end (about 0.05–0.1mm by CEJ) and becomes thicker towards the tooth apex (around 0.2–1mm). There are two types of cementum:

- acellular cementum – forms next to the root dentine during tooth development and forms around the inserting fibres of the PDL which are called *Sharpey's fibres* at this point (Fig 1-11).
- cellular cementum – contains cementocytes in lacunae and forms during function on the surface of acellular cementum.

Theoretically, the cementum should finish at the CEJ (Fig 1-12), but it can lie just

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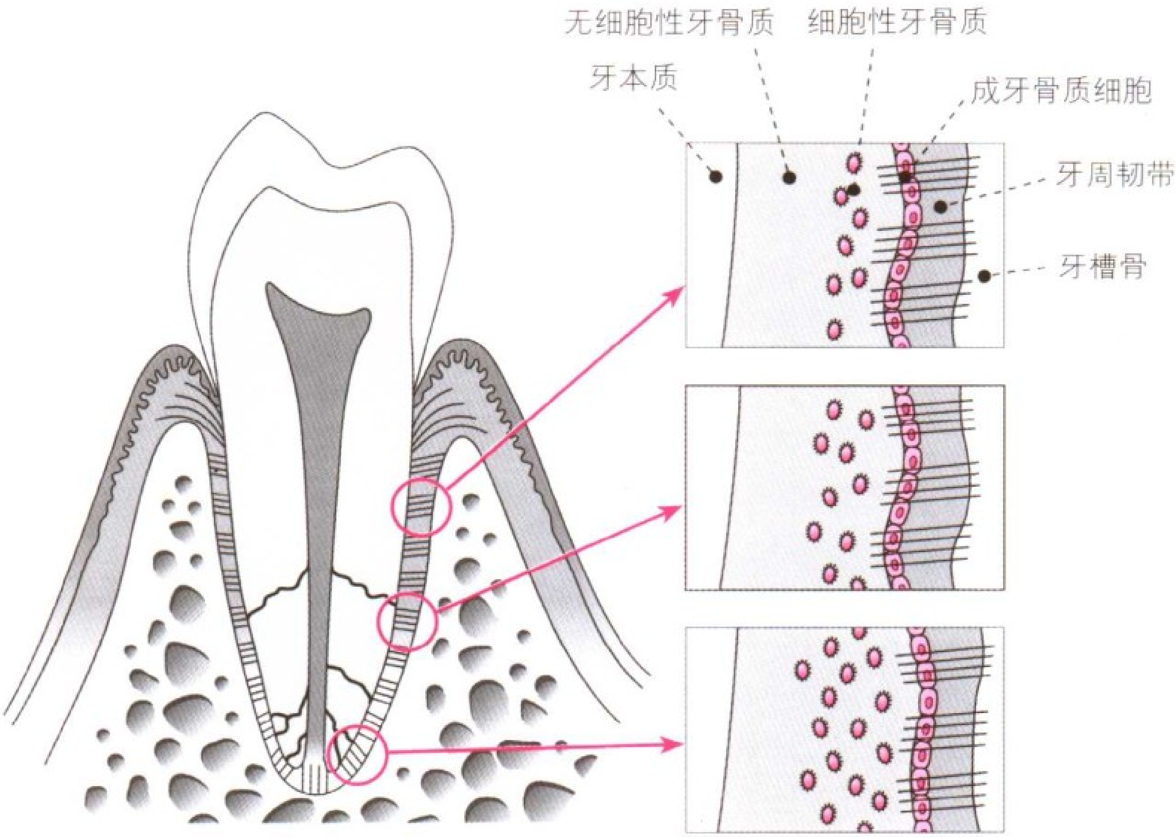


图 1-11　牙骨质结构。注意：越向根尖方向牙骨质层越厚，含牙骨质细胞越多

Fig 1-11　Schematic view of basic cementum structure. Note towards the tooth apex the cementum layer becomes thicker and more cellular.

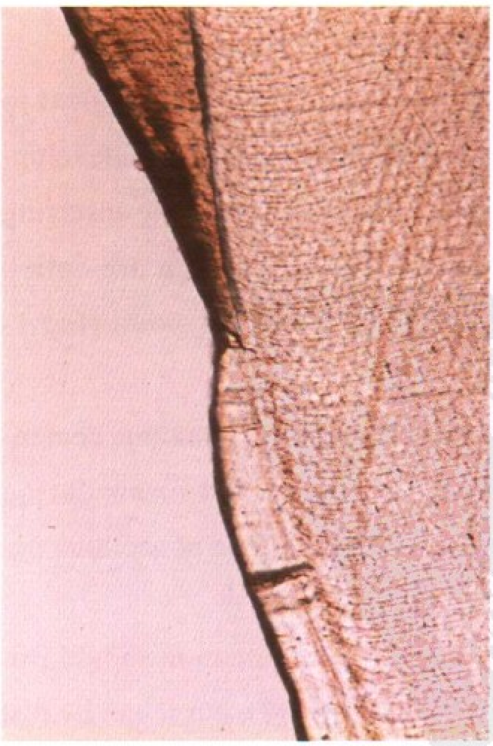


图 1-12　“正常”牙骨质与CEJ位置关系显微照片（30%）

（引自 Riviere. *Lab Manual of Normal Oral Histology*. *Quintessence*: *Chicago*, 2000）

Fig 1-12　Photomicrograph of "normal" cementum position relative to the CEJ (30% of cases).

(From Riviere. *Lab Manual of Normal Oral Histology. Quintessence: Chicago*, 2000)

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骨质珠）甚至可覆盖在牙釉质上（图1-14）。在病变牙周袋内，细菌内毒素（或脂多糖 LPS）松散地附着于牙骨质表面，根面清创术（根面刮治）(RSD）的目的是用超声波刮治器去除感染的牙骨质层（最高10%～15%）或从其表面冲洗掉细菌内毒素。

short (Fig 1-13) or have deficiencies within (cemental tears) or may even overlie the enamel (Fig 1-14). It is the surface of cementum that bacterial endotoxin (or lipopolysaccharide - LPS) loosely attaches to in the diseased pocket. Root surface debridement (RSD) aims to remove the infected cementum layer (top 10-15%) or to wash out endotoxin from its surface using ultrasonic or sonic scalers.

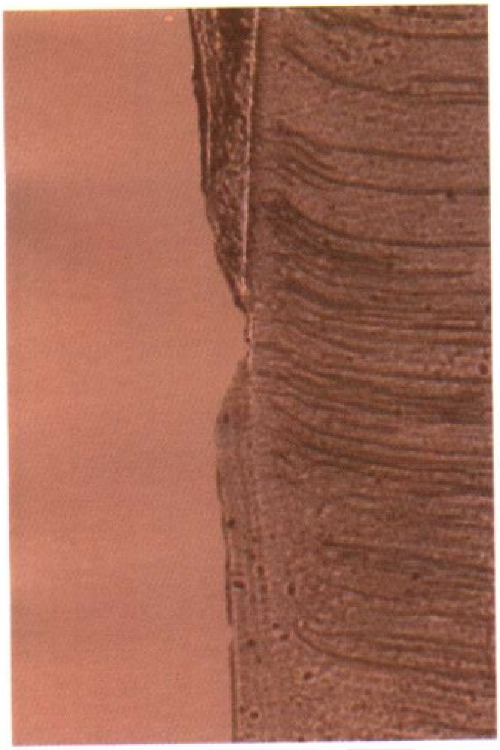


图 1-13 牙骨质位于CEJ 根方显微照片（5%～10%）

（引自 Riviere. Lab Manual of Normal Oral Histology. Quintessence: Chicago, 2000）

Fig 1-13 Photomicrograph of cementum positioned apical to the CEJ (5-10% of cases).

(From Riviere. Lab Manual of Normal Oral Histology. Quintessence: Chicago, 2000)

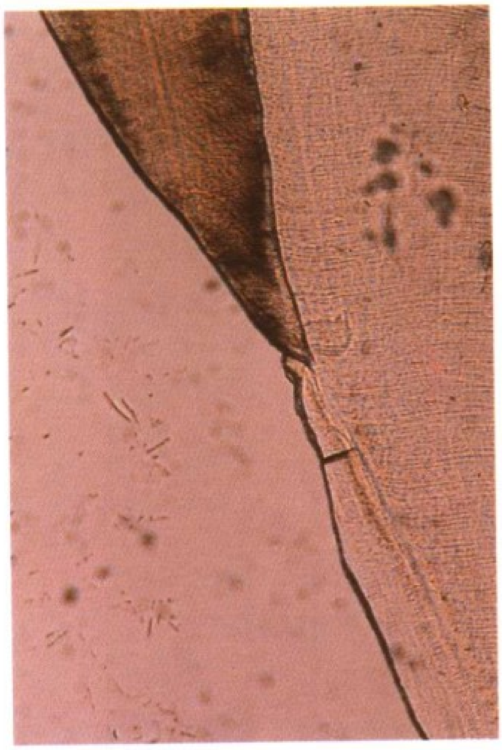


图 1-14 牙骨质位于CEJ 冠方釉质表面显微照片（60%～65%）

（引自 Riviere. Lab Manual of Normal Oral Histology. Quintessence: Chicago, 2000）

Fig 1-14 Photomicrograph of cementum positioned coronal to the CEJ, onto the enamel surface (60-65% of cases).

(From Riviere. Lab Manual of Normal Oral Histology. Quintessence: Chicago, 2000)

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牙周韧带

牙周韧带是一种比牙骨质更活跃的结构，0.2～0.4 mm宽，从CEJ根方1.5 mm处延伸并覆盖整个牙根表面，牙周韧带包括以下几种：

- 牙周韧带纤维
  - 牙槽嵴纤维
  - 水平纤维
  - 主纤维／斜行纤维
  - 根尖纤维
- 血管神经管道（通道、途径）
  - 感觉纤维（痛觉、压力和本体感觉）
  - 本体感觉纤维
  - 自主纤维
  - 血管
  - 淋巴管
- 细胞成分
  - 成纤维细胞
  - 成牙骨质细胞
  - 成骨细胞
  - 未分化的间充质细胞［常存在于牙周韧带（PDL）内，在不同刺激下能分化为任何细胞］
- 基质

牙周韧带纤维（图1-15）在液压环境中起作用，防止牙齿受到过大的殆力的损伤，组织液存在于主要的纤维束之间，而牙齿移位可刺激感觉和

PDL

The PDL is a more dynamic structure than cementum and is approximately 0.2–0.4mm wide, reaching from a point 1.5mm apical to the CEJ and investing the entire root surface. The PDL comprises the following:

- periodontal ligament fibres
  - alevolar crest fibres
  - horizontal fibres
  - principal/oblique fibres
  - apical fibres
- neurovascular channels
  - sensory fibres (pain, pressure and proprioception)
  - proprioceptive fibres
  - autonomic fibres
  - bloodvessels
  - lymphatics
- cellular elements
  - fibroblasts
  - cementoblasts/clasts
  - osteoblasts/clasts
  - undifferentiated mesenchymal cells (cells which under different stimuli can differentiate into any of the cells normally found within the PDL)
- ground substance.

The fibres of the PDL (Fig 1-15) protect the tooth from excessive forces by acting in a hydraulic environment, where tissue fluid lies between major fibre bundles and

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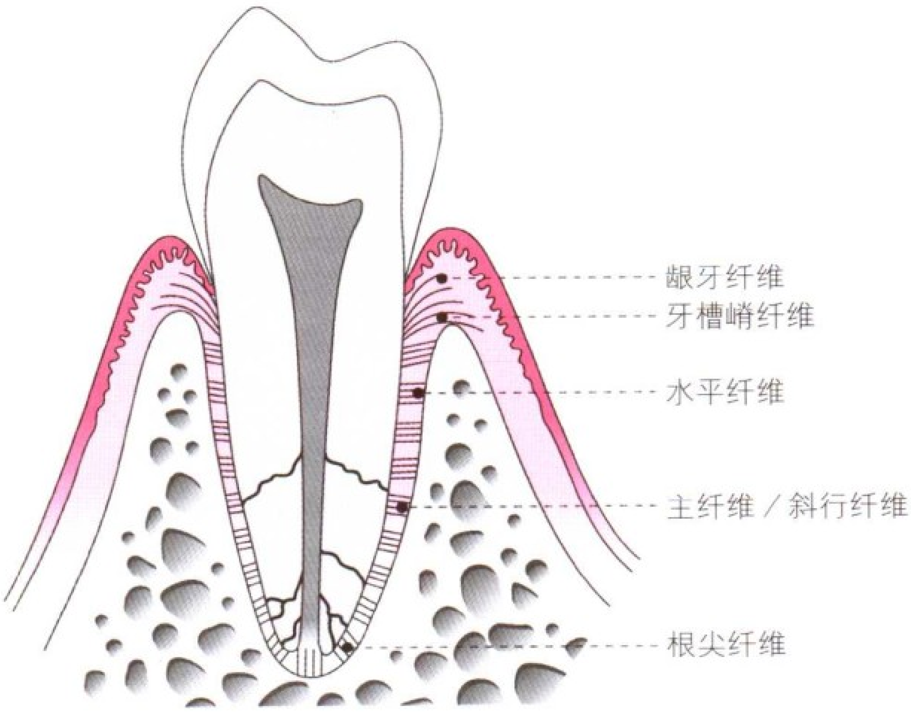


图 1-15 牙周膜纤维

Fig 1-15 Schematic diagram of PDL.

本体感觉（位置感觉）神经传导。纤维的更新速度很快且成纤维细胞非常活跃。正常情况下牙齿在牙槽窝内有极小的移动度，这称为“生理动度”（最多达 0.2 mm）。

tooth displacement stimulates sensory and proprioreceptive (positional awareness) nerve conduction. The rate of fibre turnover is high and the fibroblasts are active. Naturally the tooth can move within the socket by a small degree and this is termed *"physiological mobility"* (up to 0.2mm).

牙槽骨

上颌骨和下颌骨（向口腔内）的突出部分形成牙槽窝，称为牙槽骨或牙槽突。其在边缘较细窄，向根尖方向逐渐增厚，唇侧和舌侧致密的皮质骨板在牙槽嵴顶处相连。牙槽嵴顶正常情况下位于 CEJ 根方 1～1.5mm，在 X 线照片上形成一点（图 1-16），当牙齿间隙增宽时，牙槽嵴较为圆钝。正常情况下，皮质骨形成一条连续线，通过牙间嵴将一个牙槽窝与相邻牙槽窝相连，

Alveolar bone

The extended part of the mandible and maxilla that forms the tooth socket is called *alveolar bone* or *the alveolar process*. It is fine at its margin, becoming thicker towards the root apex, and has dense facial and lingual cortical plates, which meet at the *alveolar crest*. The latter is situated 1–1.5mm apical to the CEJ in health and radiographically forms a point (Fig 1-16), unless the teeth are widely spaced, when a more

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图 1-16 正常牙间嵴和邻牙周围的早期脱矿 X 线片

Fig 1-16 Radiograph showing healthy interdental crest with intact lamina dura and early demineralisation around adjacent tooth.

X 线片上可见一条致密的骨白线称为硬骨板。X线检查骨脱矿的第一表征就是硬骨板丧失，其常常首先出现在邻间牙槽嵴。血管、淋巴管和神经在海绵状松质骨内穿过牙槽窝的皮质骨板营养牙周韧带。皮质骨在上颌骨和下颌骨的厚度不同，在下切牙处最薄，在下颌磨牙区最厚。在下切牙区常发生发育缺陷，骨缺损处似一“窗口”，称为“开窗”。骨缺损延续致骨缘时形成“骨裂”，骨膜缺损时，该区域失去部分牙龈血管供应，可导致牙龈退缩、龈裂，伴随反复的炎症感染。此软组织裂口称为 stillman 龈裂（图 1-17）。

rounded crest may form. In health, the cortical plate forms a continuous line linking one socket via the interdental crest to adjacent sockets, visible as a fine dense white line radiographically called the *lamina dura*. The first sign of bone demineralisation radiographically is the loss of the lamina dura, which often occurs first at the interdental alveolar crest. Vascular, lymphatic and neural channels from the cancellous bone beneath perforate the cortical plate of the tooth socket, supplying the PDL. The thickness of the cortical bone varies across the maxilla and mandible, being thinnest overlying the lower incisors and thickest overlying the mandibular molars. Developmental defects can arise most commonly over lower incisors, where bone is absent as a “window” called a *fenestration*, or the fenestration communicates with the alveolar margin forming a *dehiscence*. The absence of periosteum,

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which carries part of the gingival blood supply, in these areas, can make them prone to gingival recession and cleft formation, following repeated episodes of inflammation. Such soft tissue clefts are called *Stillman's* clefts (Fig 1-17).

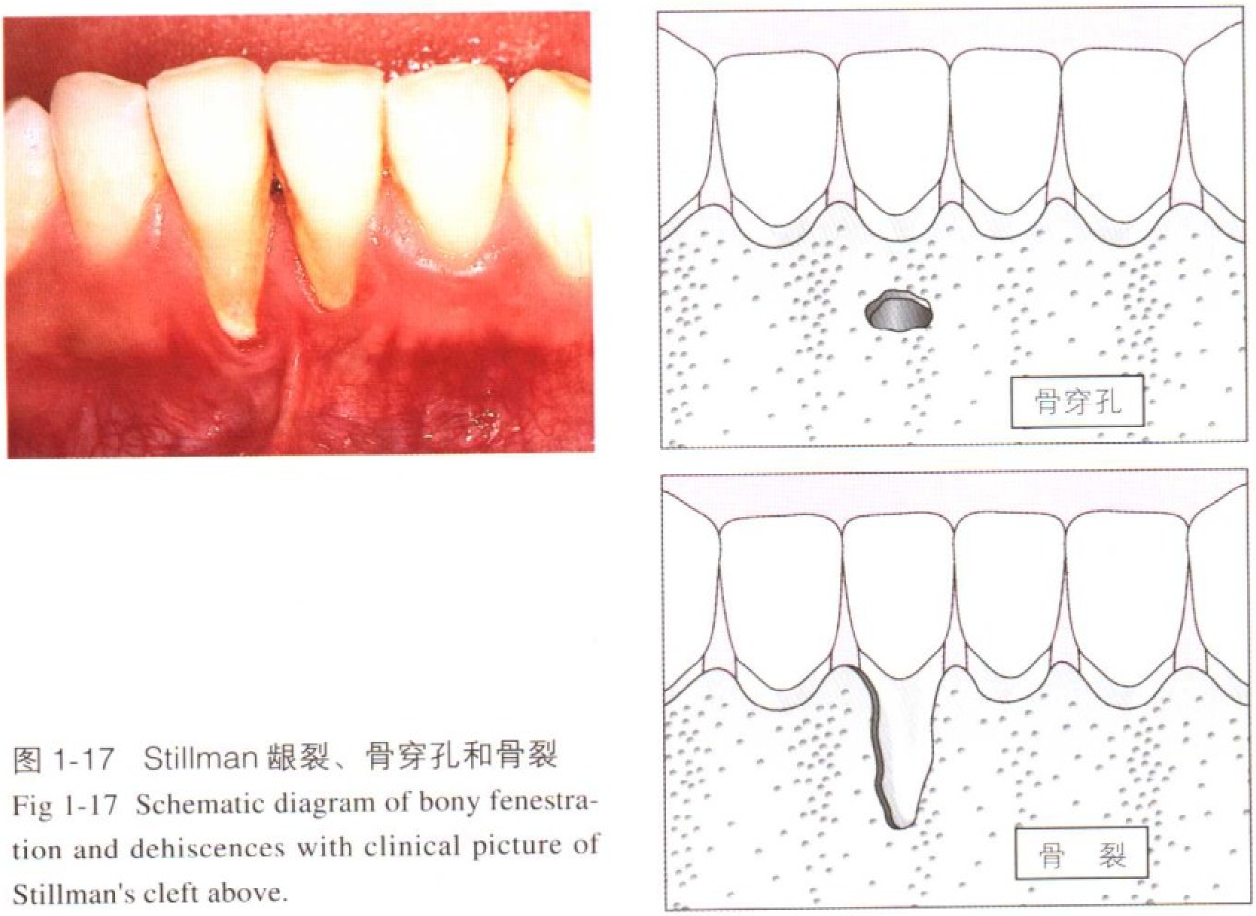


图 1-17　Stillman 龈裂、骨穿孔和骨裂
Fig 1-17 Schematic diagram of bony fenestration and dehiscences with clinical picture of Stillman's cleft above.

拓展阅读
Further Reading

Holmstrup, P. The Microanatomy of the periodontium. In Wilson Jr TG, Kornman KS (Eds.) Fundamentals of Periodontics. Chicago: Quintessence, 1996:17–45

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第 2 章 牙菌斑如何导致疾病

How Does Plaque Cause Disease

目 的

19世纪60年代的一系列研究证实了牙菌斑引起牙龈炎这一事实。牙菌斑是否引起牙周炎尚存在不同的争论。没有牙菌斑细菌，感染性牙周炎就不会发生，但当菌斑细菌存在时，也常常不引发牙周炎。本章目的在于解释在适当的环境，牙周微生物可导致易感宿主发病的机制。

要 点

通过本章的阅读可以理解当前和将来牙周治疗的微生物学基础。相对于单一感染如HIV或TB病毒感染，可以正确评价多种微生物感染的复杂性，以及为什么在牙周治疗时采用系统性抗生素对大部分患者的效果有限，而对少数患者仍有作用。

Aim

The concept that plaque causes gingivitis is a fact, proven by a series of studies in the 1960s. Whether plaque causes periodontitis is a different debate. Without plaque bacteria, inflammatory periodontitis would not occur, but in the presence of plaque bacteria, periodontitis often does not occur. This chapter aims to explain the mechanisms by which periodontal microorganisms may cause disease in a susceptible host, given the right circumstances.

Outcome

The outcome of reading this chapter will be that the reader will understand the microbial basis for contemporary and future periodontal therapies. The reader will also appreciate the complexities of a poly-microbial infection, as opposed to mono-infections like HIV or TB, and why the use of systemic antibiotics in periodontal management is of limited benefit for all but a limited number of patients.

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牙周病致病模型

单个个体牙周炎的发生发展是否取决于许多被称为“危险因素”（见第4章）的复杂问题。与大多数人都会发生的牙龈炎不同，牙周炎是由多因素引起的，不能像 HIV 或 TB 感染性疾病一样看成是单纯的传染病。它是多种微生物引起的（图2-1），受多种“宿主因素”（见第3章）和一些遗传因素、环境因素影响，并对以下临床问题有一定的影响：

A Model for Periodontal Disease Pathogenesis

Whether periodontitis develops in an individual depends upon a myriad of complex issues collectively called “risk factors” (see Chapter 4). Unlike gingivitis, which develops in the majority of, if not all, humans, periodontitis is multi-factorial and cannot be regarded as a pure infection like HIV disease or TB. It is polymicrobial (Fig 2-1) and there are many “*host factors*” (see

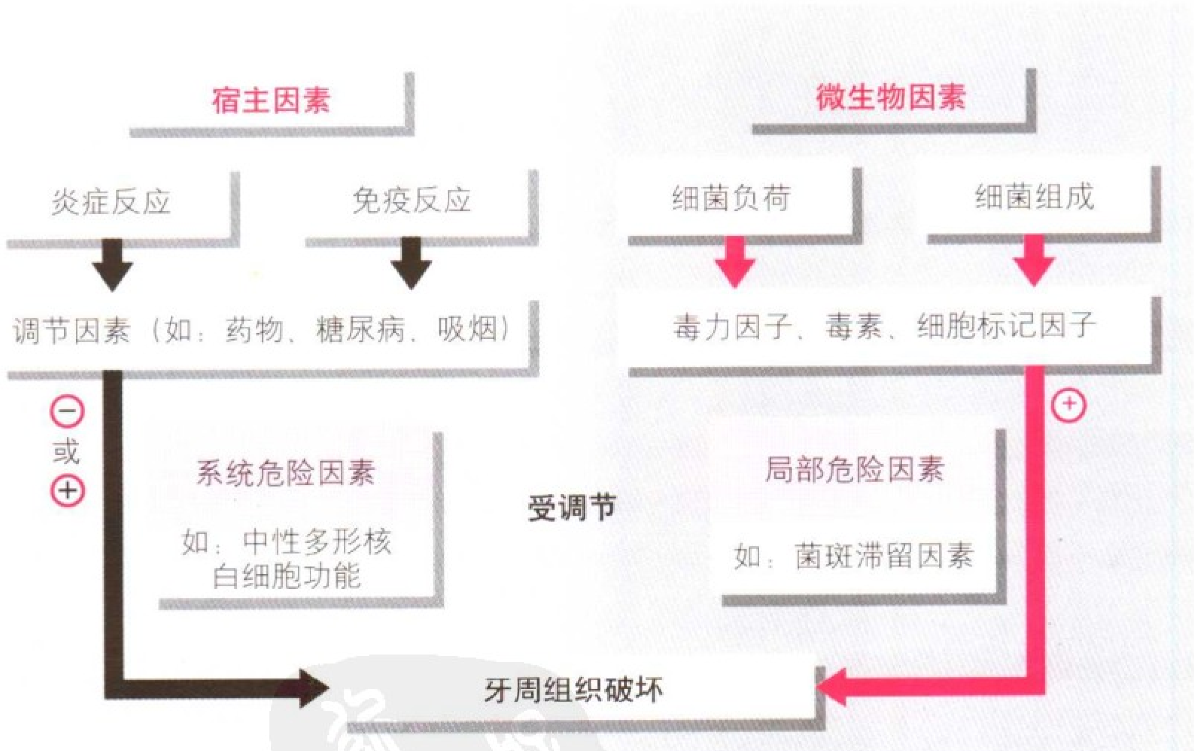


图 2-1　宿主与牙周细菌间的平衡：细菌过强的免疫反应或宿主自身炎症-免疫反应产物可导致组织破坏。个体系统水平、个体牙周位点水平存在大量促进危险因素

Fig 2-1 Schematic diagram showing how a delicate balance can exist between periodontal bacteria and the host. Tissue damage can result from bacteria overpowering the immune response or from products of the host’s own inflammatory-immune response, if this is exaggerated. On top of this are a large number of modifying or risk factors which can exist at the individual patient or systemic level, or at the individual periodontal site level (Chapter 4).

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Chapter 3), some genetically determined and some environmentally determined, which impact on the following clinical issues:

- 这个患者是否会发生牙周病？
- 如果会，将在多大年龄发生？
- 会发生什么类型的牙周病？（分类，见第6章）
- 疾病发展的速度是怎样的？
- 我们会看到什么样的疾病进展方式？（见第5章）
- 患者对治疗的反应如何？
- 什么是最恰当的治疗方法？
- 是否会造成牙齿缺失？会缺失几颗？
- 本病的过程或相应治疗会引起全身反应吗？

- Will periodontal disease develop in this patient?
- If so, at what age will it develop?
- What type of disease will develop? ("classification" – see Chapter 6)
- At what rate will the disease progress?
- What pattern of disease progression should we look for? (see Chapter 5)
- How will this patient respond to therapy?
- What is the most appropriate therapy for this case?
- Will teeth be lost? And if so, how many?
- May there be systemic consequences of the disease process or its treatment?

没有细菌就不会有牙周病，但细菌对维持口腔健康却是必不可少的，已知有超过700种细菌存在于口腔中（400～500种定植在牙周组织），只有一小部分会致病。

Without bacteria there would be no periodontal disease, but bacteria are essential to maintain health and of the 700-plus species already known to exist in the oral cavity (some 400–500 colonising the periodontal tissues), only a few cause disease.

"病原体"是一种致病的生物体。

A "pathogen" is an organism that causes disease.

"共生体"是一种不致病的生物体；是常驻菌群的一部分。

A "commensal" is a non-disease-forming organism; part of the resident flora.

"机会病原体"正常时是不致病的，一旦局部环境发生变化（如抗生素治疗减少了其竞争菌群，或宿主有免

"Opportunist pathogens" are normally not pathogenic, but are able to become so if their local environment is changed (e.g.

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疫缺陷，将会导致疾病的发生。它们能过度增生形成"微生物负荷"从而导致疾病。

by antibiotic therapy eliminating competing bacteria, or in an immunocompromised host). They can overgrow and the "microbial load" that results can cause disease.

细菌是如何致病的？

当以下基本情形出现时，细菌常导致疾病的发生：

- 获得
- 黏附
- 初步存活
- 繁殖，更长期的存活
- 避免被宿主防御系统清除
- 增殖
- 毒力因素

获得 是在出生时从父母身上（称为垂直传染）和其他环境中获得，这就有了内源性生物体（当疾病发生时内源性生物体已存在）和外源性生物体（从其他个体或环境中获得，可导致疾病或使疾病发展）的概念。1993年Van Steenbergen 用指纹遗传法证实牙周病原体如伴放线放线杆菌（*Aa*）和牙龈卟啉菌单胞菌（*Pg*）能在家族个体间传播，1988 年，Preus 和 Olsen 在一项研究中证实一名青少年与他家一只狗之间的细菌传播。然而在慢性牙周炎其致病微生物可能是内源性的，如果有外源途径，那就需要新的宿主长期暴露使细菌定植。虽然牙周病不会通过接吻传染，但我们应记住某个个体的牙周病治疗反复失败可能是由于与伙伴（伴侣）长期接触再次感染所致。

How Do Bacteria Cause Disease?

Bacteria normally cause disease when the following basic sequences occur:

- acquisition
- adherence or retention
- initial survival
- prosperity and longer-term survival
- avoidance of elimination
- multiplication
- elaboration of "virulence factors".

Acquisition of oral bacteria occurs at birth from parental (called *vertical transmission*) and other environmental sources. This has led to the concept of *endogenous organisms* (already present in the host when the disease develops) and exogenous organisms (those acquired from other individuals or the environment, which may initiate the disease process or enable its progression). In 1993, Van Steenbergen and colleagues used genetic fingerprinting methods to demonstrate that periodontal pathogens like *Actinobacillus actinomycetemcomitans* (*Aa*) and *Porphyromonas gingivalis* (*Pg*) can be transmitted between individuals within families. In one study in 1988 by Preus and Olsen, transmission between an adolescent and the family dog was

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demonstrated. However, in chronic periodontitis it is likely that causative organisms are endogenous. If exogenous sources become involved, chronic exposure is necessary for colonisation of the new host. Whilst periodontal disease is not thought to be transmitted by kissing, it is worth remembering that repeated failure of periodontal therapy in an individual may be due to re-infection from chronic contact with a partner.

黏附 是指一种微生物与组织表面的附着能力，是细菌生存最基本的要求，除非细菌是浮游的（能游动并以一种动态方式生存）。哺乳动物的牙萌出给微生物提供了一个不会脱落的稳定表面，最初黏附于牙冠表面糖蛋白薄膜（去除后数分钟内可形成）的毛物体，称为"先驱"生物体。革兰阳性球菌如链球菌通过生成黏多糖而具有此能力，而革兰阴性生物体有被称为脂磷壁酸的细胞壁成分，能促进黏附。这种机制也能帮助生物体在不需要物理附着的环境中滞留，例如未暴露在机械外力如唾液流动的聚集场所。

Adherence/retention describes the ability of an organism to attach to a surface, and is essential for bacterial survival unless the bacteria are plank-tonic (able to swim and survive in a dynamic manner). It is for this reason that the eruption of teeth in mammals provides a non-shedding, stable surface for organisms to adhere to. The first organisms to adhere to the glycoprotein pellicle that forms on crown surfaces within minutes of removal are called "pioneer" organisms. Gram-positive cocci like "streptococci" have this ability via production of mucopolysaccharides, and gram-negative organisms possess cell wall components called lipoteichoic acids, which facilitate adhesion. Such mechanisms also assist the retention of organisms in environments where physical adhesion is not an absolute requirement for colonisation, e.g. habitats not exposed to physical forces such as salivary flow.

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初步存活　不仅需要黏附于静态的表面，而且需要获得基本的营养物。细胞外基质产物（包括葡聚糖和果聚糖）促进与牙面的附着，另外黏附于龈缘和邻面的牙菌斑对口腔肌肉运动、食物颗粒及唾液流动冲刷具有较强的抗去除能力。在龈沟中，具有丰富的血清成分，包括葡萄糖（高血糖患者的龈沟内产生）和铁。然而龈沟液内由于含有铁结合蛋白如运铁蛋白、触珠蛋白和血红蛋白，以及抗体和补体，成为一种危险的环境。

Initial survival of an organism not only requires adhesion to a static surface, but also requires the acquisition of essential nutrients. The production of an extracellular matrix (comprising dextrans and levans) facilitates attachment to the tooth surface. Additionally, plaque adhering at the gingival margin and interproximally is more resistant to physical removal by the forces of the oral musculature, food particles and washing by saliva. Within the gingival crevice, GCF is rich in all serum components, including glucose (raised in GCF in hyperglycaemic individuals) and iron. The crevice is, nevertheless, a hostile environment since GCF also contains iron-binding proteins like transferrin, haptoglobin and haemoglobin, as well as antibody and complement.

繁殖和更长期的存活　需要细菌能在生物膜中与其他细菌和睦共处。生物膜是一种复杂结构，在其中有高和低的菌群区域，与水通道交织在一起。生物膜（图 2-2）的功能类似于多细胞生物体，以排出细菌表面成分（如抗原，其能激活宿主免疫反应）和释放各种毒素（如内毒素，其激活宿主炎症反应）为特征。在此环境中，细菌具有一种能动性，称为“固有指向感觉”，它只是能够对其他生物体生物膜的产物或信号，或宿主有反应，并利用它们来生长和繁殖；有的生物体与其他生物体机械性地黏附在一起（图 2-3）。

Prosperity and longer-term survival within the gingival crevice requires the bacteria to be able to survive relatively harmoniously with their neighbours in a “biofilm”. A biofilm is a complex structure within which there are areas of high and low bacterial mass, interlaced with aqueous channels. Biofilms (Fig 2-2) function like multicellular organisms characterised by shedding of bacterial surface components (e.g. antigens, which can activate a host immune response) and release of various toxins (e.g. endotoxin, which activates a host inflammatory response). Within this

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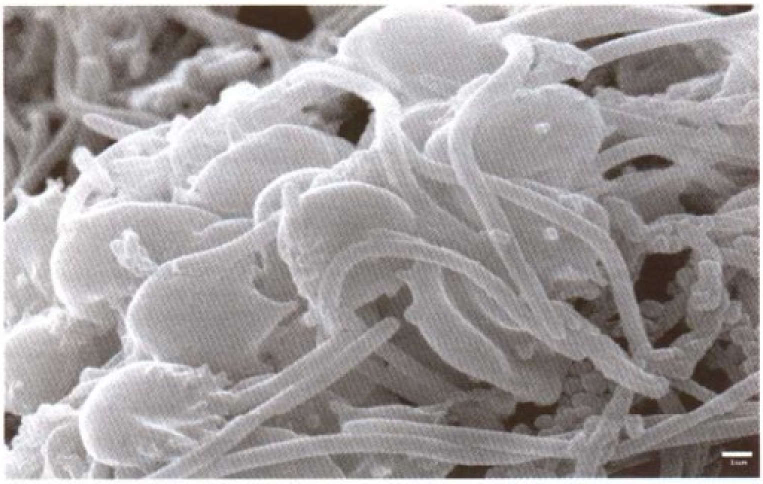


图 2-2　细菌生物膜电镜扫描照片：杆菌、球菌、丝状菌和螺旋体以及血细胞

Fig 2-2 A scanning electron micrograph (SEM) of a microbial biofilm demonstrating the intimate relationship of motile organisms with static organisms. Rod-shaped organisms can be seen along with cocci, filamantous bacteria and spirochaetes and blood cells from the host.

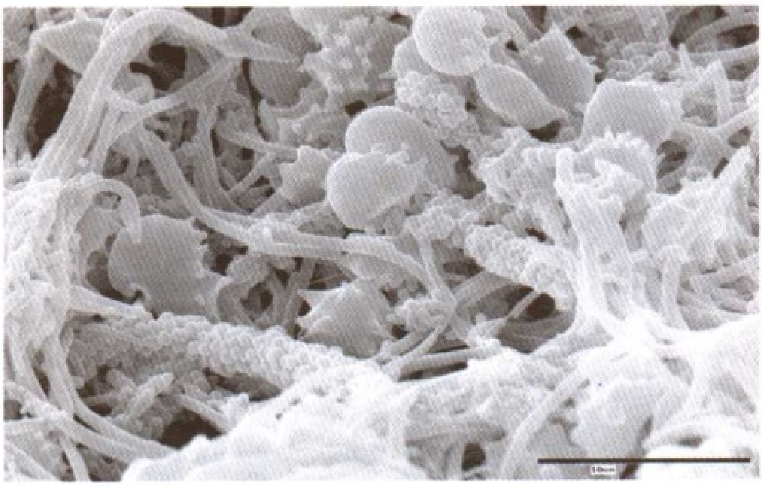


图 2-3　电镜扫描照片：球菌和丝状菌的“谷穗状”结构

Fig 2-3 SEM showing intimate relationship of bacteria within a biofilm. The alignment of cocci along the length of a filament is called a "corn-on-the-cob" relationship.

environment, bacteria are capable of an activity called "*quorum sensing*", which is simply being able to respond to products or signals from other biofilm organisms, or the host, and to use these to grow and prosper; some organisms physically adhere to each other (Fig 2-3).

避免被宿主防御系统清除　是所有病原体的主要策略，细菌被膜能促使一些生物体经历遗传“转变和漂移”并依此表面抗原进行突变，使宿主抗体不能识别和与之结合。*Aa* 产生：

Avoidance of elimination by the host defence system is a key strategy of all pathogens. Bacterial capsules can facilitate this and some organisms undergo genetic "*shift and drift*" whereby certain surface antigens mutate such that host antibodies can no longer recognise and bind to them.

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- 白细胞毒素（一种外毒素）能破坏多形核白细胞（PMNLs或中性粒细胞）
- 上皮毒素
- 免疫球蛋白酶（破坏抗体的因子）

Pg 也能产生一组蛋白酶，称为"牙龈素"，它能彻底破坏抗体、补体和其他结缔组织成分。有一些生物体能够在牙本质小管内隔离自己，使其可以在牙周袋，根面刮治后重新积聚定植（图2-4，图2-5）。慢性牙周炎用抗生素是无效的，因为此时活跃致病因素已进入生物膜，抗生素浓度太低以至于无效，而且还会出现耐药株。系统抗生素只在侵袭性牙周炎进行机械清创刮治时使用，生物膜上的生物体（细菌）对抗生素的抵抗能力是没有生物膜者的500倍，因为细胞外基质减缓了宿主防御产物的扩散，并形成一个能抑制宿主防御功能的细菌产物储存库。

Aa produces:

- *leucotoxin* (an exotoxin) that destroys polymorphonuclear leucocytes (PMNLs or neutrophils)
- *epitheliotoxin*
- *immunoglobulin proteases* (factors capable of breaking down antibody).

Pg also produces a battery of proteases called "gingipains", which can damage antibody, complement, and other connective tissue components. Some organisms are able to sequestrate themselves within dentine tubules, providing a pool for recolonisation of periodontal pockets, post-root surface debridement (Fig 2-4, Fig 2-5). The use of antibiotics is counter-productive in chronic periodontitis, because by the time the active agent has penetrated the biofilm, its concentration is too low to be effective and resistant strains of bacteria can emerge. Systemic antibiotics should only be contemplated in conjunction with mechanical debridement in aggressive forms



图2-4　电镜扫描照片：根面牙本质小管口的牙周细菌

Fig 2-4　SEM demonstrating periodontal bacteria at the entrance of dentine tubules on the root surface.

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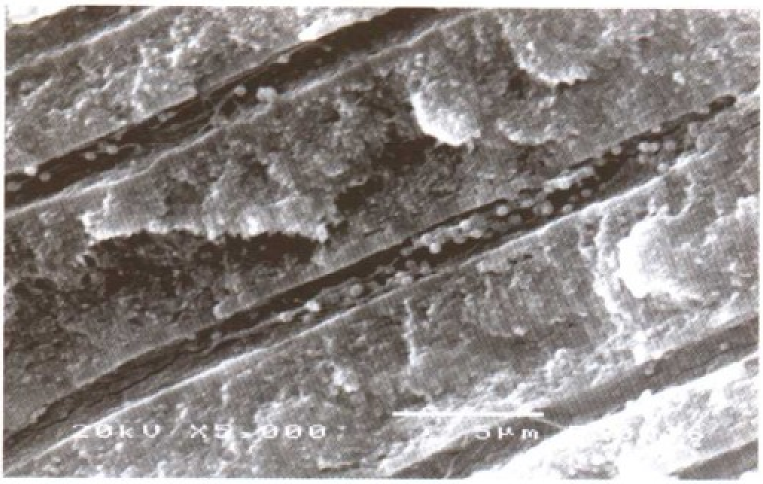


图2-5　电镜扫描照片：牙本质小管内的细菌及细胞外多糖基质产物

Fig 2-5 SEM showing bacteria within dentinal tubules with extra-cellular polysaccharide matrix production.

细菌的增殖　对于长时期的存活来说是最基本的，牙周病原微生物在龈下生物膜内需要达到一个临界聚集量才能生存。而且我们确信，超过此阈值的更大量的细菌繁殖对疾病的产生是必不可少的，但是什么能打破病原微生物的平衡使其超越临界值还不能确定：它可能是由于应急反应导致的免疫系统短暂的抑制，药物因素或其他感染（如HIV），以及微生物对宿主获取的化学药物或激素的反应能力。根据我们自己的实验室和合作实验室的研究调查，一个可能的机制是：牙周微生物（牙周致病菌）可能利用去甲肾上腺素（应激反应时产生）生成一种"自动诱导物"。此自动诱导物刺激其自身

of periodontitis. Biofilm organisms are 500-fold more resistant to antimicrobial strategies than organisms that do not occupy biofilms, because the extracellular matrix slows down diffusion of host defence products and forms a reservoir of microbial products which can inhibit host defences.

Multiplication is essential for longer-term survival and periodontal pathogens need to reach a certain critical mass within the subgingival biofilm to survive. Even once this critical mass is achieved, it is believed that further multiplication beyond a certain "threshold" is necessary for disease induction. What tips the balance for such pathogens beyond this threshold is uncertain: it may be a transient suppression of the immune system by stress, medication or other infections (e.g. HIV), or the ability of such organisms to respond to host-derived chemicals or hormones. One possible mechanism for this is under investigation in our own laboratories and those of

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生长和在病理牙周袋环境中发现的生物体的生长，且能使生物膜结构在宿主局部环境发生变化时产生转变。

collaborators, and has shown that certain periodontal organisms may use noradrenaline (produced during stress responses) to produce an "*auto-inducer*". The latter stimulates their own growth and that of other organisms found within the hostile environment of the periodontal pocket, potentially enabling shifts in biofilm composition to occur in response to changes in the local host environment.

毒力因素 是疾病产生的基本条件，生物体的毒力是指其产生疾病的能力，主要有三种毒力因素：

Elaboration of "virulence factors" is essential for the production of disease. The "virulence" of an organism describes its *power* to produce disease. Broadly there are three categories of virulence factors:

- 酶如胶原酶、透明质酸酶、弹性蛋白酶、抗补体酶、破坏抗体的酶等。这类酶已被证实能破坏上皮细胞间的结合剂（称为钙依赖蛋白分子），并能进入结缔组织，在那里它们能导致比在"外部"龈沟内可能产生的更多的破坏。

- *Enzymes* (e.g. collagenase, hyaluronidase, elastase, anti-complement enzymes, those that break antibody down and many others). Such enzymes are believed to be able to break down the epithelial inter-cellular cement (called cadherin molecules), allowing entry into the connective tissues, where they can cause more damage than is possible within the "external" gingival crevice.

- 代谢产物如氨、挥发性脂肪酸、细胞毒性胺和硫化氢（H_2S）。一些牙周病原微生物已被发现能代谢保护性的抗炎肽和抗氧化剂的肽，如谷胱甘肽并形成毒性化合物如 H_2S。

- *Metabolic waste products* such as ammonia, volatile fatty acids, cytotoxic amines and hydrogen sulphide (H_2S). Some periodontal pathogens have been shown to metabolise protective anti-inflammatory and antioxidant peptides like glutathione to form

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- 毒素包括内毒素或外毒素

– 内毒素——因其大部分只能在细菌死亡时从革兰阴性细菌的细胞壁释放而得名。但是也能在囊泡内或通过可溶解形式发生缓慢释放，已知的脂多糖（LPS）是免疫反应和炎症反应以及继而发生宿主组织破坏的最强烈刺激物之一。它们松散地附着于牙骨质上，通过超声波刮治器能轻易地去除。内毒素的其他特性包括：

- 与至多10%～15%牙骨质有松散的结合。
- 激活凝结因子（如Hageman因子），引起血管内凝结、血栓形成和牙龈局部缺血坏死。
- 激活补体（见第3章）导致炎症。
- 在酶和氧化基准备激活和释放时激活吞噬细胞。
- 激活免疫反应（抗原性）。
- 巨噬细胞毒性。

toxic compounds like H_2S.

- *Toxins* which are either *endotoxins* or *exotoxins*

– *Endotoxins* – so called, because they are only released from the cell walls of gram-negative bacteria largely when they die, but slow release can also occur within vesicles or through soluble forms. Also known as lipopolysaccharide (LPS), these are among the most potent stimulators of immune and inflammatory responses and subsequent host tissue damage. They attach loosely to cementum and are easily washed away by the irrigant from ultrasonic or sonic scalers. Other properties of endotoxins include:

- Loose association with the top 10-15% of cementum.
- Activation of clotting factors (e.g. Hageman factor) causing intravascular coagulation, thrombosis and ischaemic necrosis of areas of the gingiva.
- Complement activation (see Chapter 3) and subsequent inflammation.
- Priming of phagocytes in preparation for "activation" and release of enzymes and oxygen radicals.
- Activation of the immune response (antigenicity).
- Macrophage toxicity.

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- 骨吸收（如 *Aa* 和 *Pg*）。
- 成纤维细胞毒性。
- 抑制结缔组织附着（胶原）。

— 外毒素—由生活细菌释放，包括 *Aa* 和其他前面提到的细菌释放的白细胞毒素。

- Bone resorption (e.g. *Aa* and *Pg*).
- Fibroblast toxicity.
- Inhibition of connective tissue attachment (collagen).

— *Exotoxins* — are released by living bacteria and include the leucotoxin of *Aa* and others previously mentioned.

牙菌斑生物膜的成熟

血链球菌（一种革兰阳性链球菌）是在 24 h 内最先定植在牙冠表面的一种微生物，接着是放线菌属中的革兰阴性杆菌和丝状菌。血链球菌不是严格意义上的需氧菌或厌氧菌，已知为兼性生物体（能在有氧或无氧环境中生存）。在发生牙龈炎和龈下生物膜形成过程中牙菌斑开始成熟，菌群开始向革兰阴性菌转变，而非向革兰阳性菌转变，并变得更加能动和厌氧。牙周袋内菌群大多是可动的革兰阴性厌氧菌，且有螺旋体聚集在生物膜基底。

Maturation of the Plaque Biofilm

S. *sanguis* (a gram-positive *streptococcus*) is one of the first organisms to colonise the crown surface at 24 hours, followed by gram-positive rods and filamentous bacteria of the *Actinomyces* species. Such organisms are not strictly *aerobic* (requiring oxygen to survive) or *anaerobic* (unable to survive in oxygen), but are known as *facultative* organisms (can survive with or without oxygen). As plaque matures during the development of gingivitis and the subgingival biofilm develops, the flora shifts towards a gram-negative rather than a gram-positive flora, and becomes more motile (greater numbers of planktonic organisms) and anaerobic. The pocket flora is largely anaerobic, gram-negative and heavily motile, with spirochaetes populating the biofilm base.

细菌入侵

细菌或其产物必须进入牙周组织才能引起组织破坏。除了 1965 年 Listgarten 等证实在坏死溃疡性龈炎

Microbial Invasion

To cause tissue damage, bacteria or their products must enter the periodontal tissues. Whether periodontal bacteria physically

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(NUG)和坏死溃疡性牙周炎(NUP)(见第6章)有细菌侵入外，牙周致病菌是否在体内直接侵入宿主组织仍存在争论。一旦在龈沟上皮(SE)内形成微溃疡(见第1章)，细菌将定植于此，然而它们究竟是通过释放酶直接引起溃疡，还是由宿主炎症反应所致[由吞噬细胞通过结合上皮(JE)时释放的酶和氧自由基]，或是由两者结合所致(最有可能的情况)，我们仍然不清楚。在实验室已经证实许多病原微生物包括Pg和福赛类杆菌(Bf)能侵入宿主组织。尽管1998年Dibart等已证实在上皮细胞空泡内存在Bf(图1-7a,b)，很可能是由上皮细胞的胞饮作用所致，但在体内很难进行类似研究。

invade host tissue in *vivo* or not is controversial, with the exception of *necrotising ulcerative gingivitis* (NUG) and *periodontitis* (NUP) (see Chapter 6), where, in 1965, Listgarten and coworkers demonstrated invasion. Once micro-ulcers form within the SE (see Chapter 1), bacteria will colonise such sites. However, whether they directly cause the ulceration through release of enzymes, or whether it results from the host's inflammatory response (via enzymes and oxygen radicals released from phagocytes as they pass across the JE), or a combination of both (most likely), is unclear. *In vitro* invasion has been demonstrated for a number of pathogens including Pg and *Bacteroides forsythus* (Bf). *In vivo* invasion is, however, harder to study, and whilst Bf has been demonstrated by Dibart and colleagues (1998) inside epithelial cells (see Fig 1-7a,b), the organisms were seen inside vacuoles and are most likely to have been pinocytosed by the epithelial cells.

哪些微生物可导致牙周病？

近年来，一系列假说的提出都试图解释细菌是如何引起牙周病的。

Which Organisms Cause Periodontal Diseases?

A number of hypotheses have emerged over the years in an attempt to explain how bacteria cause periodontal diseases.

特异菌斑假说

1976年Loesche提出特异微生物是牙周疾病发生所必需的，强调的是

The specific plaque hypothesis

In 1976, Loesche proposed that specific microorganisms were necessary for the de-

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细菌的质而不是量。有证据表明Aa与局限性侵袭性牙周炎有关，Pg与弥漫性侵袭性牙周炎有关，但两种微生物都可在非疾病部位存在，在临床个别疾病也可能均不存在。对此有很多理论，最可能的解释是Aa与其他被公认的病原体有几种“克隆型”，且只有很少具有毒力，这使得在牙周疾病诊断过程中微生物的检测及识别受到局限，因为除了最复杂的检测外所有的检测对亚种均没有特异性。大多数被公认的病原体均存在于牙周袋深部的厌氧环境中，易受到由于机械操作引起的环境变化的影响。

velopment of periodontal disease, i.e. it is the quality of bacteria rather than the quantity that matters. There is good evidence to link *Aa* to localised aggressive periodontitis and *Pg* to generalised aggressive periodontitis, but both organisms are found in non-diseased sites and may also be absent when the respective diseases are present clinically. There are many reasons for this, but the most likely explanation is that *Aa* and other putative pathogens have several "*clonal types*", and only a minority are virulent. This places limitations on microbial testing and identification in the diagnosis of periodontal diseases, as all but the most complex tests are not specific to subspecies clones. The majority of these putative pathogens exist deep within the pocket in anaerobic conditions and are highly susceptible to environmental change (fragile), induced by physical instrumentation.

非特异菌斑假说

1986 年 Thielade 提出牙周疾病是由所有存在的微生物所致，一旦其超过阈值就会发病。1965 年，Löe 等设计了一系列“实验性龈炎”研究，证实菌斑诱导性龈炎适合非特异性模型。该研究让参加实验的牙学院学生让牙菌斑堆积在牙齿周围 21 d，随后发生轻微龈炎。然而菌斑堆积在已有炎症的龈缘比未感染组织更容易，且去除菌斑后龈炎消失，因而证明了“因果关

The non-specific plaque hypothesis

In 1986, Thielade proposed that the disease results from the shear mass of organisms present and once this exceeds a certain threshold, disease will occur. In 1965, Löe and co-workers performed a series of "experimental gingivitis" studies which taken together proved that plaque-induced gingivitis fitted the non-specific model. Plaque was allowed to accumulate around the teeth of dental students for 21 days and

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系"。尽管如此，有人认为缺乏由于刷牙所致牙龈血液流动的刺激，也可能引起炎症，而非菌斑的聚集所致。故该实验用氯己定消毒代替刷牙来重复进行，最后证实了这一假设关系。

mild gingivitis resulted. However, plaque accumulates more readily around the margins of gingivae that are inflamed than around un-inflamed tissues, so, to prove "cause and effect", the plaque had to be removed leading to subsequent resolution of the gingivitis. Even then, some argued that it was the lack of stimulation of gingival blood flow by brushing that may have caused the inflammation, rather than the build up of plaque, so the study had to be repeated using the antiseptic chlorhexidine, rather than brushing, finally to prove the relationship.

环境菌斑学说

1991 年 Haffajee 和他的同事描述了这一学说，环境菌斑学说提出整个龈下微生物环境对疾病的发生发展起着关键作用。消除牙周病变位点的共生细菌如血链球菌可消灭依赖血链球菌获取营养的真正致病微生物。在慢性牙周炎患者牙周袋中定植的 300～500 种微生物中，只有 10～30 种被证实是致病性的，而且在龈下生物膜中必须还有足够大量的致病微生物的存在。现在的证据证实这一学说最适合慢性牙周炎。

The environmental plaque hypothesis

Described by Haffajee and colleagues in 1991, the environmental plaque hypothesis suggests the entire subgingival microbial environment is the key to disease developing. Eliminating commensal species like *S sanguis* (Ss) from a periodontal site may also result in the elimination of true pathogens which depend upon Ss for certain nutrients. In chronic periodontitis, of the between 300 and 500 species which colonise the pocket, only 10-30 are thought to be pathogenic. Therefore, to develop periodontitis, not only is a susceptible host necessary, but pathogenic species must emerge in sufficiently high numbers from within the subgingival biofilm. Current evidence would support this hypothesis as

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the most appropriate for chronic periodontitis.

牙周袋内细菌的分层和菌群分析

在马萨诸塞州波士顿，Socransky带领一个研究小组，多年来调查研究健康和疾病状态下的牙周菌群（表2-1和

Stratification of Organisms Within the Pocket and Cluster Analysis

A group in Boston, Massachusetts lead by Socransky, has investigated over many years the periodontal microflora in health

表 2-1　牙周致病微生物和相关疾病

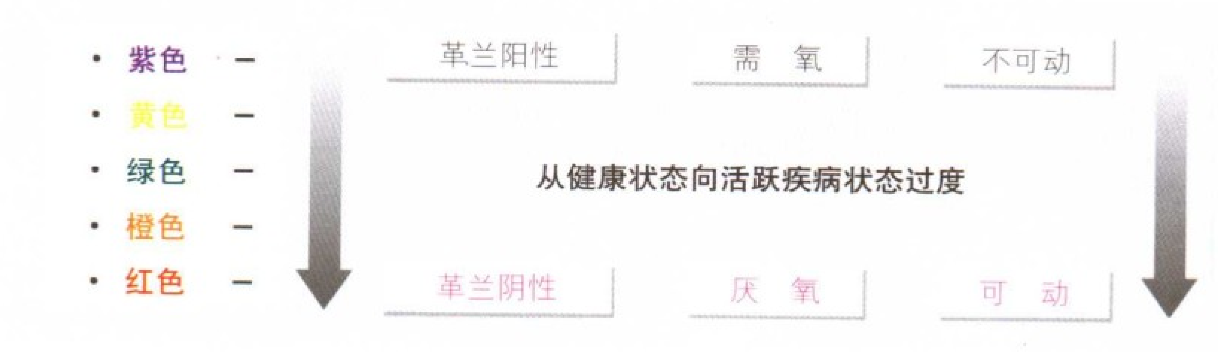
Table 2-1 Periodontal microorganisms and associated conditions.

状　况	微　生　物
健康牙龈	血链球菌 *Streptococcus sanguis*
	黏性放线菌 *Actinomyces viscosus*
	口腔链球菌 *Streptococcus oralis*
坏死溃疡性龈炎	奋森密螺旋体 *Treponema vincentii*
	具核梭杆菌 *Fusobacterium nucleatum*
	中间普氏菌 *Prevotella intermedia*
	念珠菌菌属? *Candida species?*
龈炎	具核梭杆菌 *Fusobacterium nucleatum*
	牙龈卟啉单胞菌 *Porphyromonas gingivalis*
局限性侵袭性牙周炎	伴放线放线杆菌 *Actinobacillus actinomycetemcomitans*
弥散性侵袭性牙周炎	牙龈卟啉单胞菌 *Porphyromonas gingivalis*
慢性牙周炎	牙龈卟啉单胞菌 *Porphyromonas gingivalis*
	福赛类杆菌 *Bacteroides forsythus*
	具核梭杆菌 *Fusobacterium nucleatum*
	直肠弯曲杆菌 *Camoylobacter recta*
	侵蚀艾肯杆菌属 *Eikonella corrodens*
	微小消化链球菌 *Peptostreptococcus micros*
	中间普氏菌 *Prevotella intermedia*
	真细菌属 *Eubacterium species*
	螺旋体属 *Spirochaete species*

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表 2-2）。以及牙周袋内各深度的牙周细菌的变化方式。最近对从个体牙周袋获得的大量细菌种类的研究工作证实这些细菌群聚集在独立的微环境中。这些复合菌群用颜色表示为：

and disease (Table 2-1 and 2-2) and how periodontal bacteria change along the length of a periodontal pocket. Most recently, work analysing large numbers of species from individual periodontal pockets has demonstrated that certain organisms cluster together, in discrete micro-environments. These complexes have been colour coded as:



Haffajee列举了牙周致病菌必须具备的特性：

- 必须是一种毒力克隆型。
- 必须具有致病的遗传染色体因子。
- 宿主必须对病原体易感。
- 克隆细胞系内病原体的量必须超过宿主阈值。
- 必须定植于正确的部位。
- 其他种群必须支持或至少不抑制病原体。
- 局部环境必须有助于毒力因子的产生。

Haffajee listed the properties a bacterium must possess to cause periodontitis:

- It must be of a virulent clonal type.
- It must possess the genetic chromosomal factors to initiate the disease.
- The host must be susceptible to the pathogen.
- The numbers of pathogens within a clone must exceed a threshold for that host.
- It must be located at the correct site.
- Other species must support or at least not inhibit the pathogen.
- The local environment must be conducive to the production of virulence

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表 2-2　Socransky 复合菌群

Table 2-2　Socransky's mocrobial complexes.

复合菌群	聚集的微生物
紫红色（Purple）（革兰阳性杆菌和球菌）	小费氏（韦荣）球菌 *Veillonella parvula* 溶牙放线菌 *Actinomyces odontolyticus I*
黄色（Yellow）（革兰阴性兼性球菌）	血链球菌 *Streptococcus sanguis* 铁线链球菌 *Streptococcus gordonii* 中间链球菌 *Streptococcus intermedins* 口腔链球菌 *Streptococcus oralis* 轻型链球菌 *Streptococcus mitis*
绿色（Green）（革兰阳性和革兰阴性杆菌／球菌，不可动）	侵蚀艾肯氏杆菌属 *Eikenella corrodens* 伴放线放线杆菌 血清型 a *Actinobacillus actinomycetmecomitans serotype a* 黄褐二氧化碳噬纤维菌 *Capnocytophagia ochracea* 生痰二氧化碳噬纤维菌 *Capnocytophagia sputigena* 牙龈二氧化碳噬纤维菌 *Capnocytophagia gingivalis* 伴放线放线杆菌 血清型 b *Actinobacillus actinomycetmecomitans serotype b*
橙色（Orange）（革兰阳性和革兰阳性杆菌／球菌，部分可动）	具核梭杆菌多核亚种 *Fusobacterium nucleatum ssp. polymorphum* 具核梭杆菌具核亚种 *Fusobacterium nucleatum ssp. nucleatum* 中间普氏菌 *Prevotella intermedia* 星群链球菌 *Streptococcus constellatus* 缠绕优杆菌 *Eubacterium nodatum* 纤细弯曲菌 *Campylobacter gracilis* 直肠弯曲菌 *Campylobacter rectus* 微小消化链球菌 *Peptostreptococcus micros* 变黑普氏菌 *Prevotella nigescens* 牙周梭杆菌 *Fusobacterium periodonticum* 文森氏具核梭杆菌 *Fusobacterium nucleatum ssp. vincentii* 昭和弯曲菌 *Campylobacter showae*
红色（Red）（革兰阴性厌氧菌，可动）	牙龈卟啉单胞菌 *Porphyromonas gingivalis* 福塞拟杆菌杆菌 *Bacteroides forsythus* 齿垢密螺旋体 *Treponema denticola*

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factors.

图 2-6 总结了牙周细菌在疾病发病过程中的作用。

Fig 2-6 summarises the role of periodontal bacteria in disease pathogenesis.

临床意义

- 牙龈炎是由于龈缘渐进性菌斑积聚所致的一种疾病——去除菌斑后消失。
- 大多数患者可患有牙龈炎。

Conclusions of Clinical Importance

- Gingivitis is a disease caused by the progressive accumulation of plaque at the gingival margin – plaque removal results in resolution.
- The majority of patients may suffer from gingivitis.

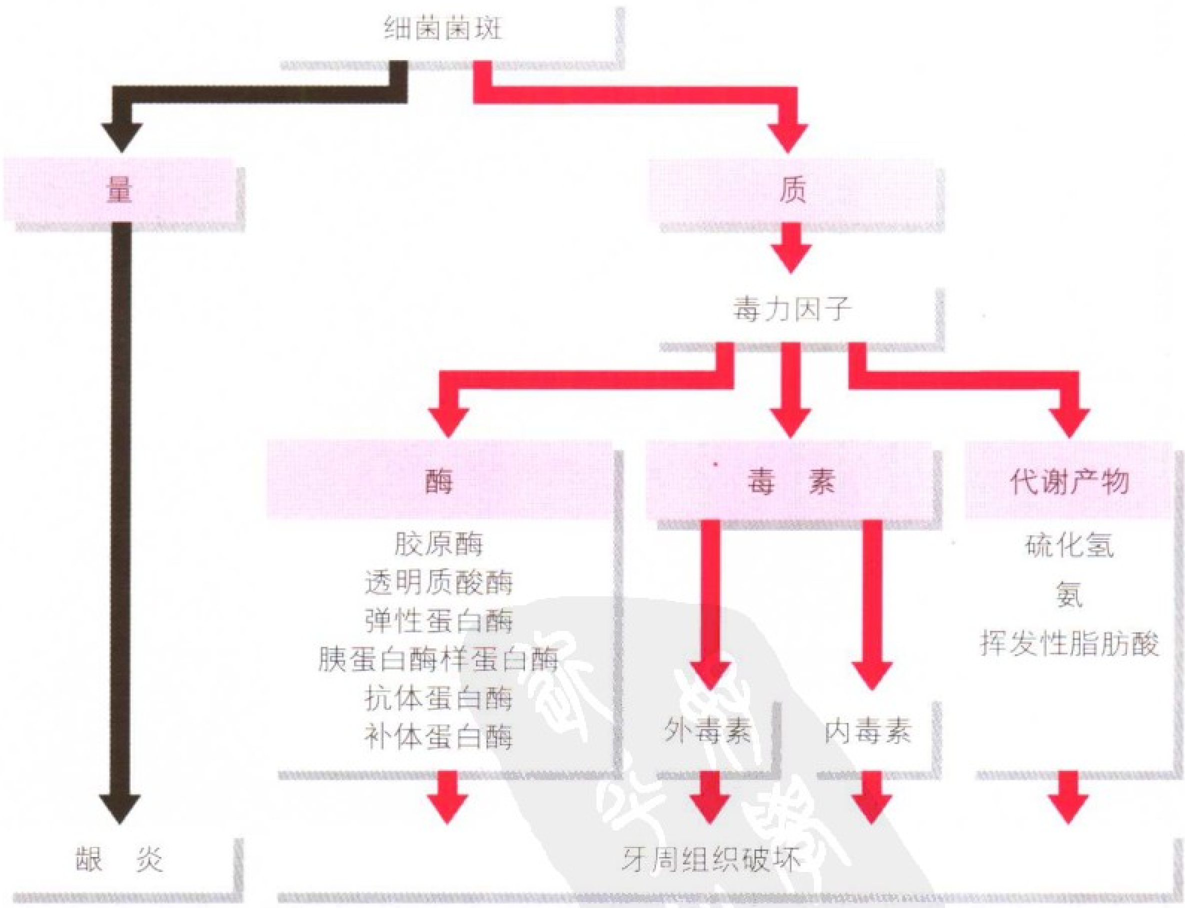


图 2-6 牙周致病微生物致组织破坏图解

Fig 2-6 Schematic representation of how periodontal micro-organisms may cause tissue damage.

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- 特异性细菌引起慢性牙周炎，它们存在于龈下生物膜，在该处细菌靠自我支持和保护能抵抗杀灭。
- 只有一小部分患者(5%～15%)对牙周炎具有易感性。
- 在治疗慢性牙周炎时不须常规使用抗生素。
- 消除脆弱的厌氧病原体首先要破坏生物膜。
- 慢性牙周炎的病原体可能是内源性的。
- 病原体在家庭内能传播。
- 治疗失败可能是由于患者特有的因素，但也可能是由于从伴侣或家庭成员中再次感染所致。
- 疾病的复发可能是由于病毒所致。

- Specific bacteria cause chronic periodontitis, but these exist in a subgingival biofilm, where self-support and protection provide resistance to elimination.
- Only a small number of patients (5%-15%) appear susceptible to periodontitis.
- Antibiotics are not normally indicated in the management of chronic periodontitis.
- Physical disruption of the biofilm is essential to eliminate fragile anaerobic pathogens.
- Pathogens in chronic periodontitis are likely to be endogenous to the host.
- Pathogens can be transmitted within families.
- Failure of therapy may be due to factors peculiar to the patient, but may also be due to re-infection from a partner or family member.
- Recurrence of disease may be due to viral organisms.

拓展阅读
Further Reading

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第3章 宿主反应的作用

The Role of the Host Response

目　的

没有一本关于牙周病学的教科书能避而不谈炎症反应和免疫反应：这些问题在牙科手术中没什么实用价值，却使许多临床操作者感到迷惑和厌烦。本章是本书最复杂的一章，力争提供一个实实在在可见的指导，以理解控制疾病发生以及治疗的主要过程。本章对理解本书后面的部分是必不可少的，同时，也必须使读者对新的诊断和治疗方法有所了解，其中一些已经受到高度重视。

Aim

No text in periodontology can avoid discussing the inflammatory and immune responses: subjects which confuse and thus bore many practitioners, often appearing to have little practical utility in the dental surgery. This chapter is the most complex in the book, but attempts to provide a substantially visual guide to key processes that govern disease development and hence therapeutic strategies. The chapter is essential to understanding the remainder of the text, and whilst contemporary, it must also prepare readers for new diagnostic and treatment strategies, some of which are already in the high street.

要　点

读完本章后读者将会理解先前认为神秘的牙周免疫病理学世界。读者将能确定新的诊断检测和治疗方法对于他们的个别患者在生物学上是有效的同时，在经济上和逻辑上是否值得。

Outcome

The outcome of reading this chapter will be that the previously mystical world of periodontal immunopathology will make some sense. The reader will also be able to make informed decisions about whether he/she feels certain newer diagnostic tests and

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therapeutic strategies, whilst biologically effective, are worthwhile, in financial and logistical terms, for their **individual** patient.

微生物菌斑引起的宿主反应是保护性的，但是低宿主反应活性或高宿主反应活性能导致组织破坏，这种平衡是微妙的（图3-1），例如图3-2中的

The host-response to microbial plaque is designed to be protective, but the balance is a delicate one where under-activity or indeed over-activity of specific aspects of

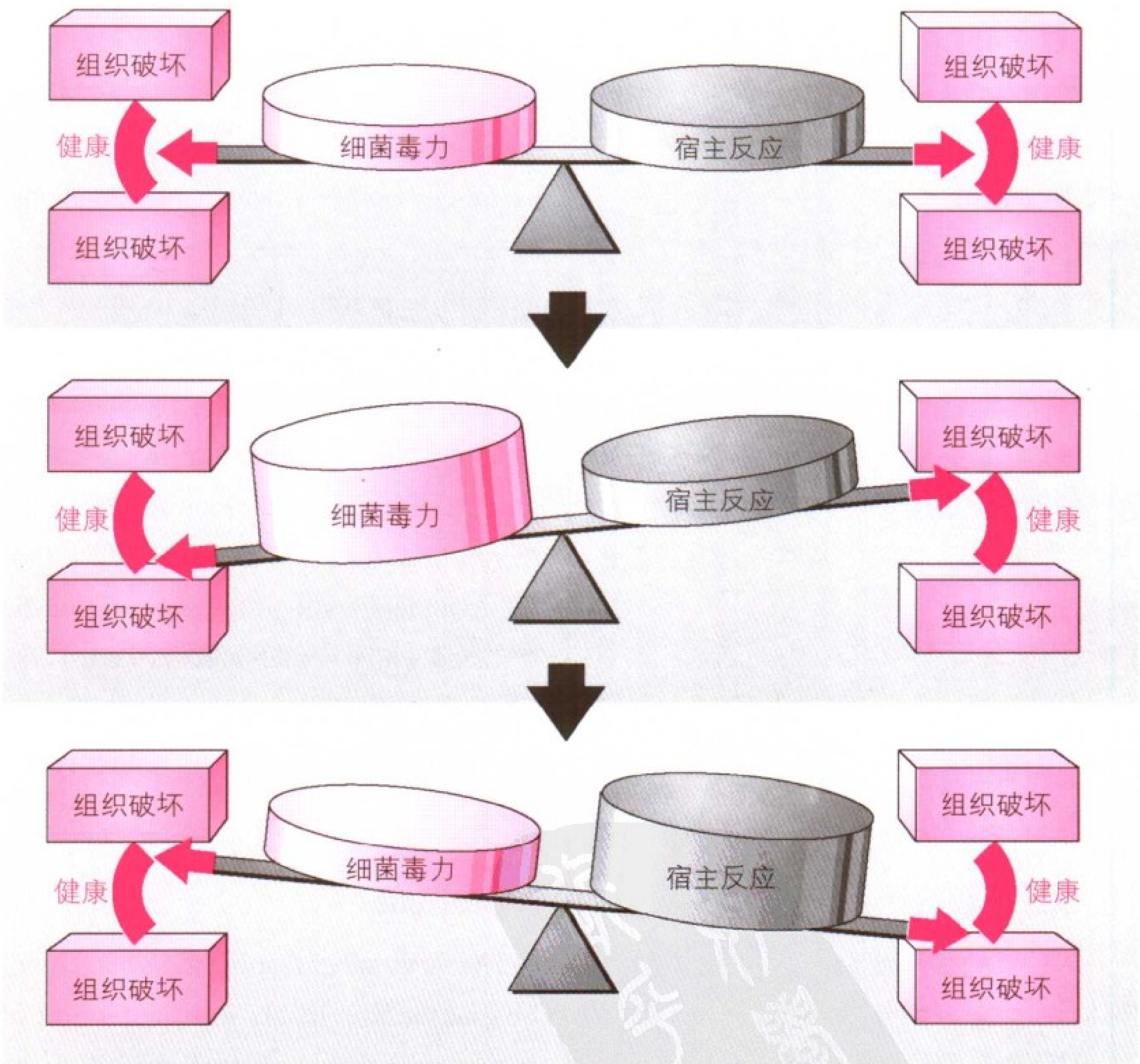


图3-1　细菌活力和宿主防御之间的平衡维持牙周组织的健康

Fig 3-1 The delicate balance between parasitic activity and host defences, which maintain periodontal tissue health. Such equilibrium may easily be disrupted, leading to inflammation and tissue damage.

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图 3-2　口腔卫生良好的 C1– 酯酶抑制因子功能异常患者炎症反应严重

Fig 3-2 Clinical photograph of exaggerated inflammation in a patient with excellent oral hygiene, who suffers from C1-esterase inhibitor dysfunction.

患者患有 C1– 酯酶抑制剂功能缺陷，这种酶能阻止补体 C1（能使炎症过程蔓延）的激活。当牙菌斑在患者口内龈缘聚集时，导致的炎症和肿胀过大，并能引起严重的牙龈水肿和骨吸收。菌斑引起的宿主反应主要有两种：

- 自然或先天免疫性
- 后天或特异免疫性

宿主对细菌产生的免疫反应包括以上两种类型，并同时出现，但是先天免疫在功能上长期稳定且能更迅速地动员起来，而后天免疫系统涉及淋巴细胞和特异细胞间的相互作用，需更多的时间。早期牙龈炎症主要涉及先天免疫系统，而中度至重度牙龈炎和牙周炎则涉及后天免疫系统（即长期、慢性疾病）。

the response can lead to tissue destruction (Fig 3-1). For example, the patient in Fig 3-2 suffers from the defective function of an enzyme called C1-esterase inhibitor. The enzyme inhibits the activation of complement component C1 (which propagates inflammatory processes). When plaque accumulates at the gingival margin in her mouth, the resulting inflammation and swelling are exaggerated and cause severe gingival oedema and bone loss. There are two main components of the host-response to plaque:

- natural or innate immunity
- acquired or specific immunity.

Both types of immunity are involved in the host's response to plaque and occur at the same time, but the innate system is constantly in function and more rapidly mobilisable, whereas the acquired system involves lymphocytes and specific cell–cell interactions which take more time. Early gingival inflammation largely involves the innate system, the acquired sys-

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tem becoming involved in moderate-to-advanced gingivitis and in periodontitis (i.e. longer-term, chronic disease).

先天免疫

先天免疫系统是宿主防御的前线。通过进化，先天免疫系统更加稳定迅速，相对于后天免疫系统的特性，其作用是无选择性的。在牙周免疫学方面来说，类似于用大锤击打一颗小钉子，所以当这一系统出现问题时，附属组织的破坏显著。先天免疫包括：

- 完整的上皮屏障
- 含抗菌因子的液体（唾液、龈沟液）对上皮的润滑
- 补体级联
- 细胞因子（如白介素家族）和趋化因子（由一个细胞产生的分子，吸收有特异性白细胞）
- 由肥大细胞释放的血管活性肽（如组胺）
- 黏附分子
- 中性多形核白细胞（中性粒细胞）防御系统
- 巨噬细胞——在特异性免疫反应开始时作为抗原呈递细胞（APCs）行使功能

Innate Immunity

This system is the first line of defence. Throughout evolution, it has been highly conserved, and, whilst rapid, it remains crudely indiscriminate relative to the specificity of the acquired system. In terms of periodontal immunology, it is akin to hitting a small nail with a sledgehammer, and hence, when something goes wrong with this system, the *collateral tissue damage* is significant. Innate immunity involves:

- intact epithelial barriers
- lubrication of epithelium with fluids (saliva, GCF) containing anti-bacterial factors
- the complement cascade
- cell-signalling molecules called *cytokines* (e.g. the interleukin family) and *chemokines* (molecules produced by one cell, which recruit specific leucocytes)
- vasoactive peptides (like histamine) released from mast cells
- adhesion molecules
- PMNL (neutrophil) defence systems
- macrophages – which also function as *antigen presenting cells* (APCs) at the beginning of specific immune

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responses.

上皮屏障

第1章中我们已讨论了上皮屏障。结合上皮的独特性提供了基本的一线防御，结合上皮能够快速更新，且对龈沟液和中性多形核白细胞具有渗透性。上皮细胞也释放细胞标记分子（详见下文）促使炎症过程的发生。

Epithelial Barriers

Epithelial barriers were discussed in Chapter 1. The unique nature of the JE, its rapid rate of turnover and permeability to GCF and PMNLs passing through, provide primary first-line defence strategies. The epithelial cells also release cell-signalling molecules (see below) to help start the inflammatory process.

液体润滑

总之，混合唾液含有可以与细菌相结合的抗菌酶（如溶菌酶）和免疫球蛋白（抗体）如IgA和IgC。龈沟液中携带有血清的所有成分，包括补体和免疫球蛋白。

Fluid Lubrication

Whole, mixed saliva contains anti-bacterial enzymes, like lysozyme, and immunoglobulins (antibody) like IgA and IgG, which bind to bacteria. GCF carries all components of serum, including complement and immunoglobulin.

补体级联

补体级联是20种血清糖蛋白系列，在血液中以非活跃的形式循环。当被激活后，补体成分具有强烈的刺激炎症的效应（炎症前效应）。补体激活的主要作用有：

- 吸引更多的吞噬细胞到感染区域
- 促进吞噬细胞（如中性多形核白细胞）与细菌结合，帮助吞噬作

Complement Cascade

The complement cascade is a series of 20 serum glycoproteins, which circulate in inactive forms in the blood stream. When activated, complement components have profound and powerful effects in stimulating inflammation (pro-inflammatory effects). The main role of complement activation is:

- recruitment of more phagocytes to the area of infection
- to facilitate binding of phagocytes (e.g. PMNLs) to bacteria, thereby aiding

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用——这一过程称为调理素作用

- 引发灭菌（细胞溶解）

补体激活有两种途径（图 3-3）：

- 旁路途径——细菌内毒素（LPS）由牙骨质储库进入牙周组织直接激活。其他蛋白（水解）酶如胞浆素和一些神经肽如应激过程中释放的 β-内啡肽也能激活旁路途径。

phagocytosis – a process called *opsonisation*

- to cause bacterial killing (cell lysis).

There are two pathways for complement activation (Fig 3-3):

- *alternative pathway* – activated directly by bacterial endotoxin (LPS) when it enters the periodontal tissues and from the cementum reservoir. Other proteolytic enzymes like plasmin and some neuropeptides (e.g. β-endorphin, released during stress

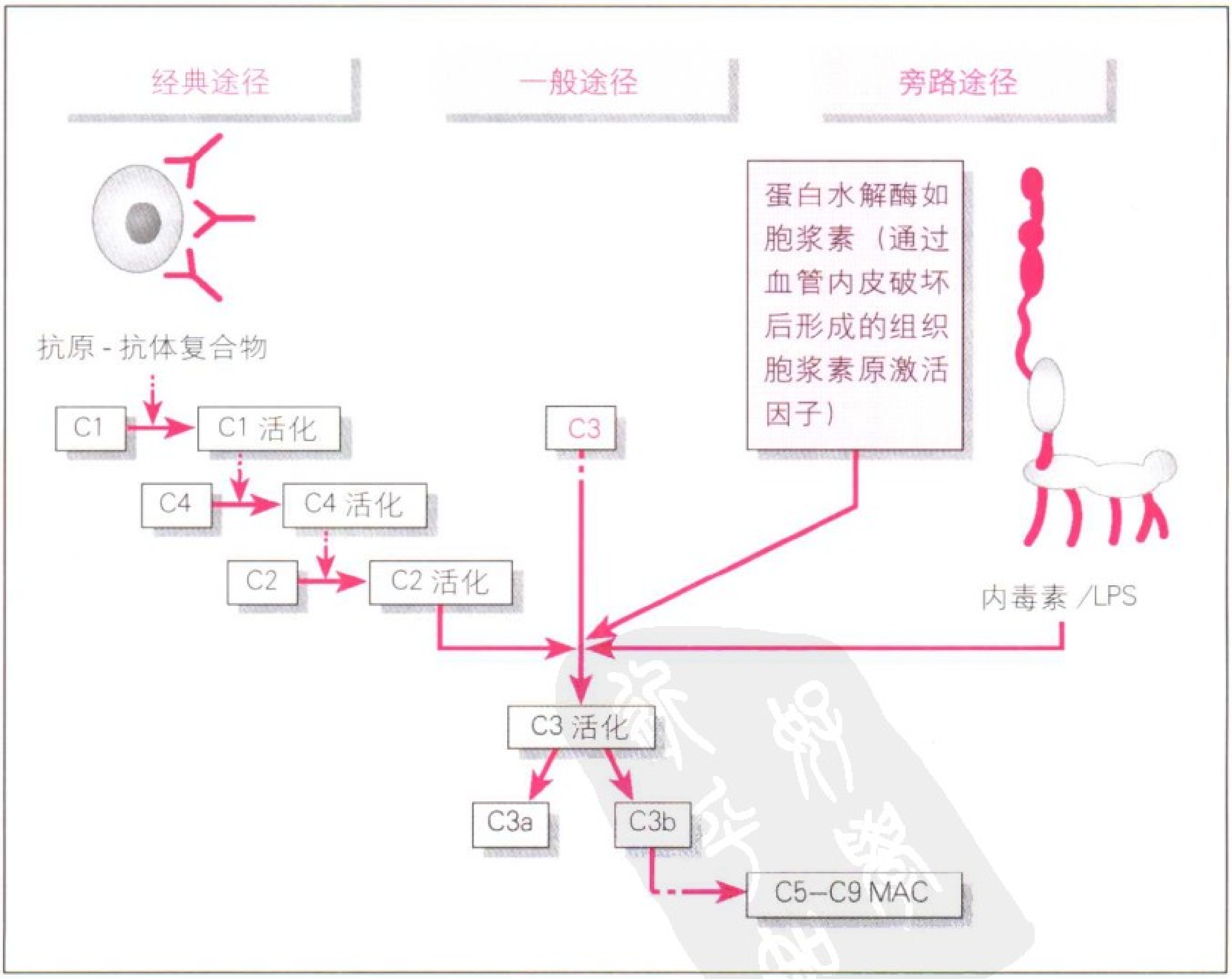


图 3-3　补体级联

Fig 3-3 The complement cascade.

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responses) can also activate the alternative pathway.

• 经典途径——只被抗原—抗体合成物激活（例如获得性免疫反应发生后）

• *classical pathway* – only activated by the formation of antigen-antibody complexes (i.e. after initiation of the acquired immune response).

两种途径都能刺激补体 C3 的激活，补体 C3 导致 C5 的激活和级联放大，并形成 C5～C9，即所谓的膜侵袭合成物（MAC）。C3～C5 的活动型吸引吞噬细胞，使炎症扩散的方法归纳见表 3-1。可看到内毒素能够直接激活这一补体级联，通过补体C3b，介导中性多形核白细胞与细菌的天然依附（调理素作用），接着中性多形核白细胞通过细胞外或细胞内杀灭机制杀灭细菌（详见下文），这可能没有包含淋巴细

Both pathways stimulate the activation of complement component C3, which leads to the activation of C5 and the amplification cascade which forms C5-C9, the so-called *membrane attack complex* (MAC). The methods by which active forms of C3 to C9 recruit phagocytes and propagate inflammation are summarised in Table 3-1. It can be seen how endotoxin can directly activate this cascade and, via complement component C3b, mediate the crude adher-

表 3-1　补体的重要效应

Table 3-1 Some important effects of complement.

补体成分	炎　症　效　应
C3a 和 C5a	通过巨细胞脱颗粒和释放组胺使血管通透性增加
C3b 和 C5a	白细胞产生氧自由基
C5a	白细胞附壁于血管壁
	中性粒细胞释放酶和氧自由基（称为脱颗粒）
C3a	吞噬细胞趋化作用（化学吸引）
C3b	生成细胞因子（淋巴细胞产生）
	分泌巨噬细胞
	调理素作用（吞噬细胞的吸引和黏附）
C5a 和 C567 复合物	白细胞趋化作用（向炎症移动）
C5b，C6，C7，C8 和 C9	革兰阴性细菌溶解
	宿主细胞分解破坏

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胞和特异性免疫反应。这种反应类型被确认为早期龈炎所具备。

ence of PMNLs to bacteria (opsonisation). PMNL-killing of the bacteria then follows through *extracellular* or *intracellular* killing mechanisms (see later), which may not involve lymphocytes and specific immunity. This type of action is believed to be responsible for early gingivitis.

细胞标记分子

存在多种此类分子家族：有的刺激细胞释放其他分子（细胞因子），有的吸引细胞到感染区域（趋化因子），有的刺激细胞完成其他功能（细胞因子和淋巴因子）。这些高度复杂的网状系统的功能和动力学还没有被认识清楚，许多还有似乎相对的活性，大多数则具有普通活性。牙周炎症过程中主要的细胞因子见表3-2。在牙周病中最重要的细胞因子之一是白细胞介素1（IL-1），它是一种强烈的炎症前介质，能导致骨吸收。编码IL-1的基因具有不同形式（多态性），kornman等发现重度慢性牙周炎患者具有IL-1基因多态性，因此提出了"牙周炎易感性检测试验"（PST），通过采集指血样本来识别"危险"基因多态性的存在。但是，患者的吸烟习惯使该结论变得混乱不清，没有明确证据表明PST阳性意味着患者将发生牙周病。另外，在美国进行研究的同时，在英国同时进行的一项试图证明同一结论的实验失败了。

Cell-signalling Molecules

Several families of these potent molecules exist; some stimulate cells to release other molecules (cytokines), some attract cells to areas of infection (chemokines) and others stimulate cells to perform other functions (cytokines and lymphokines). The function and dynamics of these highly complex networks are poorly understood and many have seemingly conflicting activities, whilst others have common activities. The main cytokines and their functions in periodontal inflammation are listed in Table 3-2. One of the most important cytokines in periodontal disease is interleukin-1 (IL-1), as it causes bone resorption and is powerfully pro-inflammatory. The genes that code for IL-1 have different forms (polymorphisms) and Kornman and colleagues found that patients who suffered from advanced chronic periodontal disease possessed a polymorphism of IL-1 that resulted in overproduction. A "periodontitis susceptibility test" (PST) has been developed from this work, which identifies from a finger-

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表 3-2　一些重要的细胞因子及其作用
Table 3-2 Some important cytokines and their effects.

细胞因子	细 胞 来 源	炎症／免疫反应
IL-1	巨噬细胞（MΦ） 成纤维细胞 单核细胞（由内／外毒素刺激） 上皮细胞	破骨细胞激活 ↑ PMNL 附壁（着边） ↑ 牙周成纤维细胞释放前到腺素 PGE_2 ↑ 牙周成纤维细胞合成 IL-6 ↑ INF-α 的产生和释放
IL-6	巨噬细胞 成纤维细胞 上皮细胞	↑ 急性期蛋白合成（肝脏） ↑ 骨吸收 ↑ B 细胞分化和免疫球蛋白产生 ↑ T 细胞的激活
IL-8	巨噬细胞 内皮／上皮细胞 被刺激的 LPS 成纤维细胞，血小板	强烈的 PMNL 趋化作用
IL-10	T 淋巴细胞 B 淋巴细胞	细胞因子抑制（抗炎症的）
TGF-β	多数细胞	抗炎（高浓度） 刺激胶原合成和修复
TNF-α	活化的巨噬细胞 单核细胞 上皮细胞	↑ 巨噬细胞产生 IL-1 ↑ 巨噬细胞产生 PGE-2 ↑ ICAM-1 表达 /PMNL 附壁 ↑ PMNL 氧自由基产生 ↑ PMNL 酶的脱颗粒
PGE_2	活化的巨噬细胞 单核细胞 PMNLs 巨细胞 上皮细胞	↑ 血管通透性 ↑ 血管舒张 ↑ PMNL— 趋化作用 刺激骨吸收
GM-CSF	上皮细胞 单核细胞 内皮细胞 成纤维细胞	↑ 骨髓释放 PMNLs ↑ PMNL 趋化作用 ↓ 细胞凋亡 ↑ 氧自由基形成

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prick blood sample the presence of the "risk" polymorphism. However, patients' smoking habits confound the results, and there is no clear evidence that a PST-positive result will mean the patient concerned will develop periodontal disease. In addition, a study in the UK failed to demonstrate the same results as the United States study in a UK cohort.

血管活性肽

血管活性肽如组胺，在炎症的发展中起着决定性的作用，组胺是在刺激（如补体C3a、C5a或PGE2）存在时由肥大细胞释放，并导致血管舒张使更多的血液细胞和血浆蛋白（如补体，抗体）进入感染区。血管舒张同时使血管内血液流速减慢，使PMNLs接触血管壁而不是在血流中心（详见下文），组胺同时提高了血管通透性，使血浆中的防御产物进入组织。主要的吞噬细胞为中性粒细胞和单核细胞，当它们进入组织后变为巨噬细胞或郎格汉斯细胞（上皮巨噬细胞）。图3-4描述了早期血管变化时细胞因子和血管活性肽的作用，其能使炎症进展。

Vasoactive Peptides

Vasoactive peptides, like histamine, play a crucial role in the development of inflammation. Histamine is released from mast cells upon stimulation (e.g. by complement C3a and C5a or PGE2) and causes *vasodilation* to bring more blood cells and plasma proteins (e.g. complement, antibody) to the area of infection. Vasodilation also slows down blood flow through vessels, allowing PMNLs to touch the vessel walls, rather than being carried midstream within blood (see below). Histamine also increases *vascular permeability* to allow defence products within plasma to enter the tissues. The main phagocytes are neutrophils (PMNLs) and blood monocytes, which when they enter tissues become macrophages or Langerhans cells (epithelial macrophages). Fig 3-4 illustrates the role of cytokines and vasoactive peptides in the early vascular changes that enable inflammation to progress.

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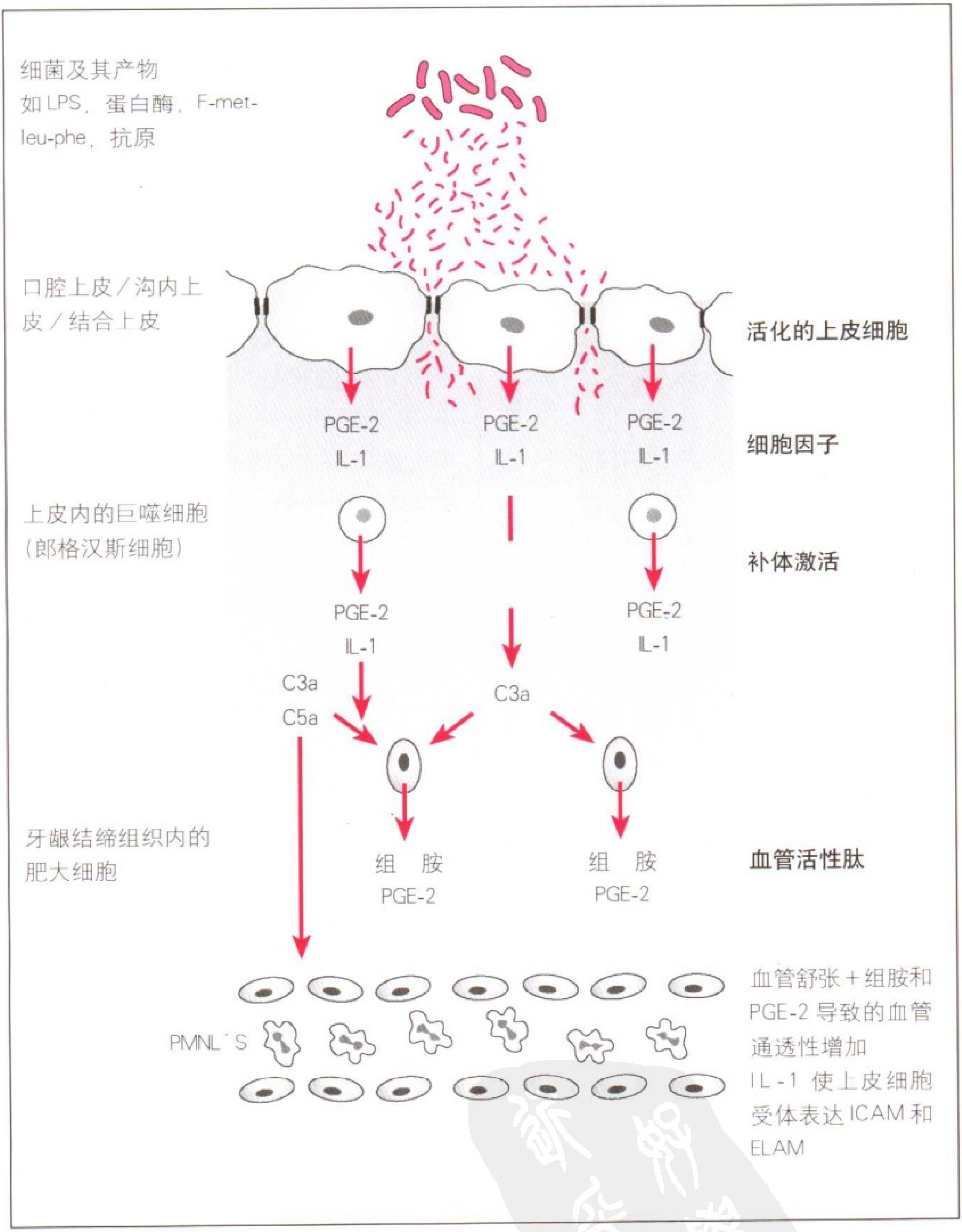


图3-4 上皮细胞、肥大细胞、巨噬细胞和补体在引起早期炎症主要血管变化的细胞因子和血管活性肽产生中的作用

Fig 3-4 Schematic representation of the role of epithelial cells, mast cells, macrophages and complement in the production of cytokines and vasoactive peptides that produce key vascular changes in early inflammation.

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黏附分子

顾名思义，黏附分子是宿主细胞用以黏附其他宿主细胞或细胞间基质的。不同的描述名称包括：黏附分子；受体（例如整联蛋白或选择蛋白），配体等。理解先天性免疫要掌握的关键概念是细胞通过和进入组织不是一个随意的过程：它是由信号分子和黏附分子以特定顺序和途径来介导。重要的黏附分子有细胞内黏附分子Ⅰ和Ⅱ（ICAM-Ⅰ and Ⅱ），内皮黏附分子1（ELAM-I）和白细胞功能抗原-1（LFA-1）。这些分子在或不在细胞表面表达，表达能被细胞因子（如IL-1，IL-10）打开或关闭。图3-5描述了在黏附分子表达时细胞因子的作用以及它们与PMNL受体黏附时引起的以下结果：

- 滚动——在血液中由于血管舒张，PMNLs流动减慢，随着血管活性肽的释放。在PMNL受体（称选择蛋白）与血管内皮上的补体间的接触可“形成与打破”。

Adhesion Molecules

As the name suggests, adhesion molecules are used by host cells to stick to each other or to components of the intercellular matrix. Various terms are used to describe such molecules: for example, adhesion molecules, receptors (e.g. integrins or selectins), ligands, etc. The key concept to grasp when attempting to understand innate immunity is that cellular trafficking into and within the tissues is not a random process: it is directed by signalling molecules and adhesion molecules in very specific sequences and pathways. Examples of important adhesion molecules are intercellular adhesion molecules I and II (ICAM-I and II), endothelial adhesion molecule 1 (ELAM-I) and leucocyte function antigen-1 (LFA-1). These molecules may or may not be expressed on a cell surface, and expression can be turned on or off by cytokines (e.g. IL-1, IL-10). Fig 3-5 illustrates the role of cytokines in the expression of adhesion molecules and how these stick to PMNL receptors to cause the following sequence of events:

- *Rolling* – slowing down of PMNLs within the blood stream occurs due to vasodilation, following the release of vasoactive peptides. “Make and break” contacts between PMNL receptors (called selectins) and complementary

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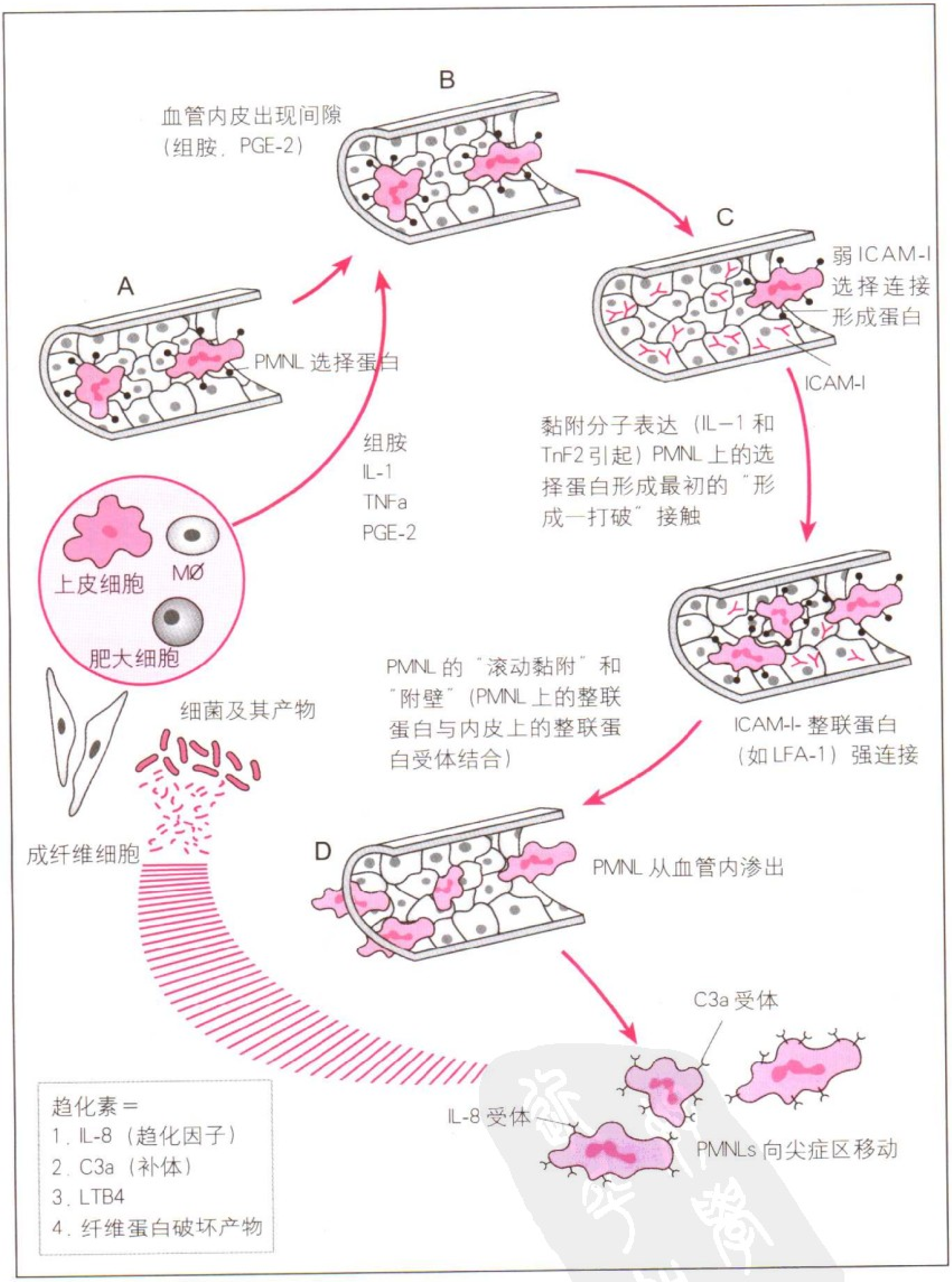


图 3-5　血管内中性粒细胞向炎症部位聚集

Fig 3-5　Schematic representation of the processes involved in the recruitment of neutrophils from blood vessels to sites of tissue damage and infection.

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• 附壁——当PMNL的流动减慢，受体的连接变得更强，由于整联蛋白分子（如LFA-1）与补体内皮受体（如ICAM-Ⅰ）的黏附使血管内皮上的PMNL固定。

• 白细胞渗出——其他细胞与细胞黏附允许PMNL通过"漏的"血管壁（组胺所致）进入组织。

• 趋化作用——PMNL受到来自细菌或宿主的趋化素的诱导，沿化学梯度进行移动。

多形核白细胞（或中性粒细胞/PMNL）

PMNL是牙周组织中最丰富和重要的防御细胞，甚至在临床健康状态，也能发现PMNL渗出牙龈内血管，通过结缔组织进入龈沟，最后进入唾液。中性粒细胞的预期寿命只有24 h，具有4种主要的功能成分：

• 受体——存在于PMNL表面，与许多不同刺激（物）发生反应，一些帮助它结合到血管壁（例如整联蛋白LFA-1）；或识别化学吸

ones on vascular endothelial cells are then able to occur.

• *Margination* – as the PMNL slows down the receptor binding becomes stronger and the PMNL becomes immobilised on the vascular endothelium by adhesion of *integrin* molecules (e.g. LFA-1) with complementary endothelial receptors (e.g. ICAM-I)

• *Diapedesis* – Other cell-to-cell adhesions allow the PMNL to pass through the "leaky" vessel wall (leaky due to histamine, etc.) and enter the tissues.

• *Chemotaxis* – The PMNL now moves along a chemical gradient, attracted by chemotaxins, which may be of bacterial or host origin.

The Polymorphonuclear Leucocyte (or Neutrophil/PMNL)

The PMNL is the most abundant and important defence cell in the periodontal tissues. Even in clinical health, PMNLs are found passing out of blood vessels within the gingiva, and through the connective tissues into the gingival crevice and ultimately saliva. Neutrophils have a short life-expectancy of only 24 hours and have four main components of functional importance:

• *Receptors* – on the PMNL surface respond to many different stimuli. Some help it to bind to the vessel wall (e.g. integrins like LFA-1); or recognise

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引剂（例如趋化素受体C3a或IL-8）；其他则帮助PMNL通过调理素作用（例如补体C3b受体或抗体受体——Fc受体）与细胞黏附。

- 细胞支架——显然，为了移动，在PMNL内有一个内在支架，由肌动蛋白肌纤维构成（肌动蛋白细胞支架）。
- 溶酶体——也称为中性白细胞颗粒。有原发性、继发性和非特异性颗粒，它含有破坏细菌结构的酶（例如 PMNL 弹性蛋白酶、组织蛋白酶、磷酸酶、胶原酶、脂酶等）。
- 氧自由基——PMNL含有一个特殊的"径路"称为NADPH氧化酶或己糖－单磷酸盐分路。它存在于PMNL细胞内膜上，简单地将己糖和NADPH从产能（ATP）循环中分流出来，减少氧以形成氧自由基（所谓的呼吸爆发）。这些包括过氧化物和相关种类，如过氧化氢和羟基，它们能破坏细菌组织和宿主组织。

一旦PMNL到达炎症部位，即通过细胞内或细胞外途径杀灭细菌。除

chemical attractants (e.g. receptors for chemotaxins like C3a or IL-8); others help it stick to bacteria through opsonisation (e.g. receptors for complement C3b or antibody receptors called Fc-receptors).

- *Cytoskeleton* – Clearly, in order to move, the PMNL has within it an internal skeleton made of actin muscle fibres (*actin cytoskeleton*).
- *Lysosomes* – are also called neutrophil *granules*. There are primary, secondary and non-specific granules, which essentially contain the enzymes used to destroy bacterial structures (e.g. PMNL elastase, cathepsins, phosphatases, collagenases, lipases, etc.)
- *Oxygen Radicals* – The PMNL contains a special "shunt" called the NADPH-oxidase or hexose-monophosphate shunt. This is situated on the inner PMNL-cell membrane and simply shunts hexose and NADPH out of the energy- (ATP-) producing cycle, and uses them to reduce oxygen to form oxygen radicals (the so-called respiratory burst). These include *superoxide* and related species like *hydrogen peroxide* and *hydroxyl radicals*, which destroy bacterial (and host) tissues.

Once the PMNL arrives at the site of infection, it kills bacteria by either *intrac-*

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PMNL在细胞内和在特殊的膜结合结构内（图3-6）安全地分泌酶和氧自由基

ellular or *extracellular* methods. Intracellular killing involves exactly the same

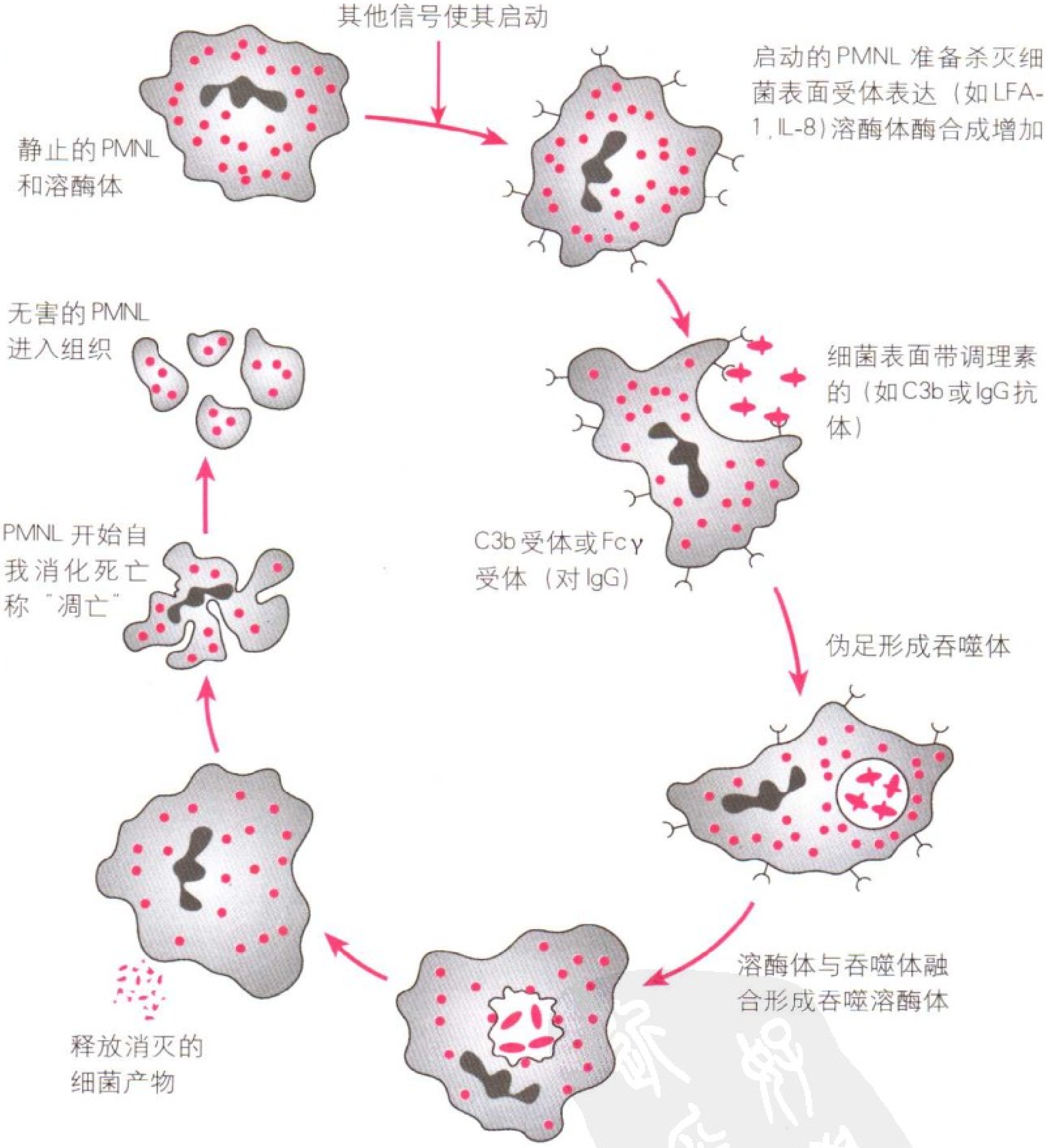


图3-6　中性粒细胞细胞内的杀灭机制

Fig 3-6 Schematic representation of the processes involved in neutrophil killing by intracellular mechanisms.

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（一个称为脱颗粒的过程）外，细胞内杀灭细菌的方法与细胞外途径相同。显然，这些酶和氧自由基也可像破坏细菌一样破坏宿主组织。故 PMNL 含有：

methods as extracellular killing, except the PMNL releases its enzymes and oxygen radicals (a process called degranulation) safely within itself and inside special membrane-bound structures (see Fig 3-6). Clearly, the enzymes and oxygen radicals may also damage host tissue as well as bacterial tissue and the PMNL therefore contains:

- 酶抑制剂——主要有 α -1 抗胰蛋白酶（像弹性蛋白酶一样的中性酶）。
- 抗氧化剂——它们是氧自由基的强力清除剂（例如谷胱甘肽），也包含酶系统（例如超氧化物歧化酶和过氧化氢酶），以防止氧自由基释放过程中破坏 PMNL。

- *enzyme inhibitors* – the main one being a-1 antitrypsin (neutralises enzymes like elastase)
- *antioxidants* – these are powerful scavengers of oxygen radicals (e.g. glutathione) and also involve enzyme systems (e.g. superoxide dismutase and catalase) to prevent damage to the PMNL during oxygen radical release.

随着细菌的细胞内杀伤，PMNL 经历了一个程序性的细胞死亡或细胞凋亡过程，靠它自我消化来防止有害成分进入宿主组织。有些病原体如*Pg*，产生抗凋亡因子，现已确信其能引起不受控制的 PMNL 死亡，随后出现 PMNL 介导的宿主组织破坏。

Following intracellular killing of bacteria, the PMNL goes through a process of programmed cell death or *apoptosis*, whereby it essentially self-digests to prevent harmful contents entering the host tissues. Some pathogens like *Pg* produce anti-apoptosis factors believed to allow uncontrolled PMNL death and subsequently PMNL-mediated host tissue damage.

当细菌团块过大，PMNL 不能进行吞噬时（图 3-7），它进行细胞外脱颗粒并释放酶和氧自由基到细菌团块表面，尽可能地破坏细菌团块，如果这一过程在龈沟内出现，就可能破坏龈沟

When the bacterial mass is too large for the PMNL to phagocytose (Fig 3-7), it degranulates extracellularly and releases its enzymes and oxygen radicals over the bacterial mass in an effort to cause as much

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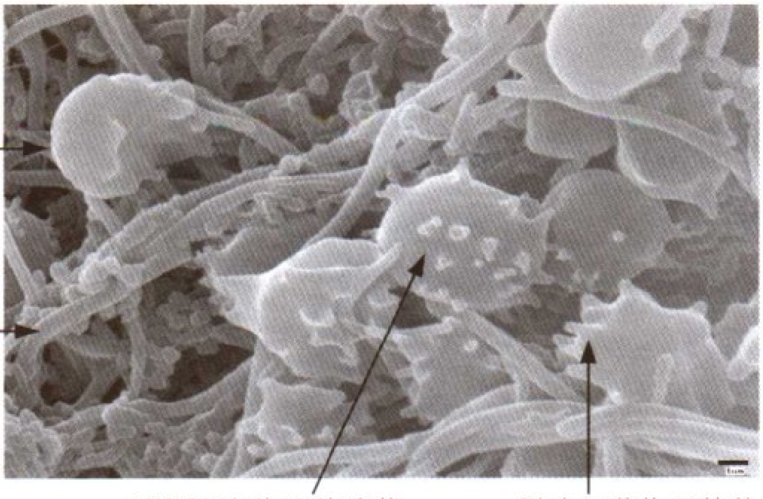


图 3-7　龈下菌斑电镜扫描照片，显示调理球菌的 PMNLs，部分 PMNLs 由于细菌团块过大而出现细胞外脱颗粒

Fig 3-7 SEM of a subgingival plaque sample, demonstrating cocci opsonised to PMNLs and active phagocytosis. Some PMNLs have degranulated extracellularly, due to the mass of bacteria being too large to engulf.

上皮，在组织内大量的PMNL反应出现的地方，不经意释放的这些化学物质被认为是造成牙周组织破坏和骨吸收的主要原因。

damage as possible . If this occurs in the gingival crevice, it may damage crevicular epithelium, but where a massive PMNL response is present in the tissues, inadvertent release of these chemicals is believed to be the major cause of periodontal tissue damage and bone loss.

巨噬细胞（MΦ）

巨噬细胞是死亡细胞（细菌或PMNLs）的清除剂，而且在先天免疫和后天免疫之间起着重要的桥梁作用。当吞噬细胞行使与PMNLs相同形式的功能时，它们表现也类似抗原呈递细胞（APCs）。巨噬细胞像其他所有人类细胞（红细胞除外）一样有同样的表面糖蛋白，称为MHC-抗原（MHC=主要组织相容性复合物；也称HLA或人类

Macrophages (MΦ)

Macrophages are scavengers of dead cells (bacterial or PMNLs), but also play an important role in bridging the gap between *innate immunity* and *acquired immunity*. As phagocytes, they function in a similar manner to PMNLs, but they also act *as antigen presenting cells* (APCs). Macrophages possess the same surface glycoproteins as all other human cells (except

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白细胞抗原)。MHC-抗原有两种主要类型即Ⅰ型和Ⅱ型，MHC-Ⅱ型是能够使宿主免疫系统识别“宿主组织”与“外来组织”的抗原系统。当巨噬细胞经过它们不能识别的细胞时，就会吞噬这些细胞，并从与MHC-Ⅱ型分子相连接的细胞中释放抗原物质到细胞表面。对外来物质具有特异性的T淋巴细胞识别这一MHC-Ⅱ型抗原/外来抗原复合体，并与之结合，进而继续发动免疫反应。同时当出现T细胞抗原表达时，MΦ释放IL-1和TNF-α，它们具有先前所述的血管效应(图 3-4)。所以，MΦ在先天免疫和获得性(特异性)免疫中都具有作用。MΦs产生引起骨吸收的PGE_2。Offenbacher等已经发现当给予同样的刺激时，从牙周病患者外周血中采集的单核细胞(血液MΦ)比健康对照组产生更多的PGE_2。我们不知道这是否是由于疾病组中的MΦ已结合了血液中的内毒素或是疾病易感个体固有地产生更多的PGE_2所致。

red blood cells) called MHC-antigens (MHC = major histocompatibility complex; also called HLA or Human Leucocyte Antigens). There are two main types of MHC-antigens (class I and class II) and MHC-class II is the antigen system that enables the host's immune system to recognise “host” from “foreign” tissue. When macrophages come across cells they do not recognise, they phagocytose them and release onto their surface antigenic material from those cells that is linked to MHC-class II molecules. T-lymphocytes, already specific to the foreign material, recognise this complex of MHC-class II/foreign antigen, bind to it and then go on to mount a specific immune response. At the same time as antigen presentation to T-cells, the MΦ releases IL-1 and TNF-α, which have the vascular effects described previously (Fig 3-4). Hence, the MΦ has a role in innate immunity and also in stimulating acquired/specific immunity. MΦs produce PGE_2, which causes bone resorption. Offenbacher and colleagues have found that monocytes (blood MΦs) collected from peripheral blood of periodontitis patients produce considerably more PGE_2 than those from healthy controls, when given the same stimulus. Whether this is because the MΦs in the diseased group were already *“primed”* by blood-borne endotoxin or whether disease-

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susceptible subjects produce inherently more PGE_2 is unknown.

获得性／特异性免疫

这最后一部分将对获得性免疫给出一个简明的总结。重要的是记住先天免疫反应与后天免疫是同时出现，但比后天（获得性）免疫的特异性和有效性要低。获得性免疫包括：

– T淋巴细胞
– B淋巴细胞
– 免疫球蛋白

T淋巴细胞 构成细胞介导的免疫反应，因为T细胞上的抗原特异分子（T细胞受体，TCR）并不是T细胞分泌的，而是黏附在T细胞表面。所以要摧毁细菌T细胞必须和细菌的直接接触，就是说这个反应是由T细胞本身而不是由血清中的自由抗体"介导"。

B淋巴细胞 构成体液免疫反应，因为B细胞产生抗原特异性分子（免疫球蛋白—Igs）。当B细胞分泌抗原特异性分子的时候它们将变成浆细胞。这些抗原特异性分子由浆细胞分泌，并进入组织液（体液）和血液中。免疫球蛋白在补体和吞噬细胞的帮助下，引起特异性的细菌的破坏。

Acquired/Specific Immunity

This final section will provide a brief overview of acquired immunity. It is important to remember that the innate immune response is occurring at the same time as the acquired response, but is less specific and thus less efficient. The acquired response involves:

- *T-lymphocytes*
- *B-lymphocytes*
- *Immunoglobulins.*

T-lymphocytes make up the *cell-mediated* immune response, so-called because the antigen-specific molecule on the T-cell (the T-cell receptor [TCR]) is not secreted/released from the cell, but remains stuck to the T-cell surface. Thus, direct contact between the T-cell and bacteria is necessary to cause bacterial destruction, i.e. the response is "mediated" by the T-cell itself and not by free antibody within serum.

B-lymphocytes form the *humoral immune* response, so-called because B-cells produce antigen-specific molecules (immunoglobulins – Igs). When this occurs they become plasma cells. The antigen-specific molecules are released from the plasma cell and enter tissue fluid (humor) and blood. Igs, with the help of complement and phagocytes, cause destruction of bacteria

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免疫球蛋白——有五种主要类型，称为 IgG、IgA、IgM、IgD 和 IgE（抗体/免疫球蛋白）。在牙周病中最重要的是 IgG（来自龈沟液）和 IgA（来自唾液）。

免疫球蛋白

在任何种感染中，第一个产生的免疫球蛋白都是 IgM，因为 IgM 有 5 个免疫球蛋白单位（即是说，它是五邻体）和交键分子井。IgG是后来产生的，由于它的分子量小（单体）和普遍存在，大概是最重要的抗体类型。IgG 从血浆中渗出进入龈沟，随后通过龈沟液进入唾液中。唾液免疫球蛋白属于 IgA 类型，它是以二聚物的形式被释放，随着腺体分泌单位（称为一个"J-链"）的增加，在唾液中出现。单体的免疫球蛋白的基本结构如图 3-8 所示。这种免疫球蛋白分子有四条链，两条轻链在末端与抗原结合 [FAB（抗原结合片断）]，两条重链从FAB末端延伸至

to which they are specific.

Immunoglobulins – There are five main classes, called IgG, IgA, IgM, IgD, IgE *(antibody/Igs)*. The most important in periodontal disease are IgG (from GCF) and IgA (from saliva).

Immunoglobulins

The first immunoglobulin to be produced in any infection is IgM, because it'has five immunoglobulin units (i.e. it is a pentomer) and cross-links molecules well. IgG is produced later and is probably the most important antibody type, owing to its small molecular weight (monomeric) and ubiquity. IgG enters the gingival crevice and subsequently saliva from blood plasma, via GCF. Salivary immunoglobulin is of the IgA type, which is released as a dimer and emerges within saliva following the addition of a secretory unit (called a"J-chain!") by the gland. The basic structure of monomeric immunoglobulins is shown in Fig

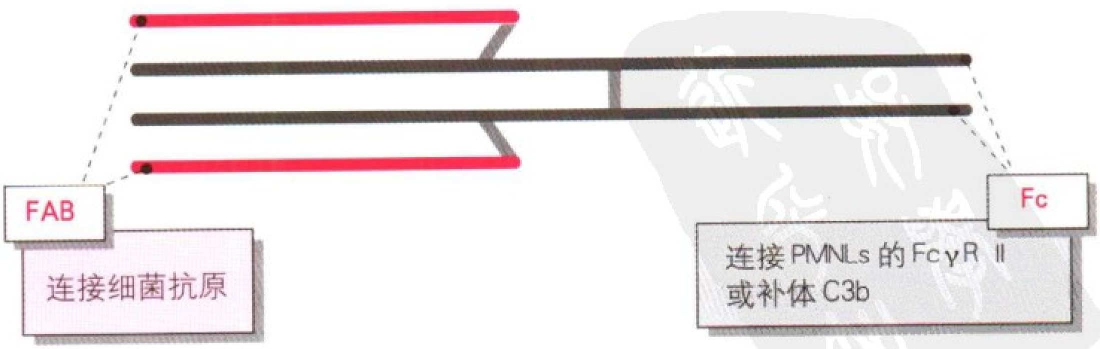


图 3-8 免疫球蛋白结构

Fig 3-8 The structure of immunoglobulin.

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结合PMNL-受体的末端［Fc（片断是恒定的）］。IgM和IgG激活（固定）补体；IgG存在四种类型，称为IgG1、IgG2、IgG3或IgG4，IgG2激活补体的作用很弱，所以细菌杀伤作用也弱，IgG2也是一种弱调理素（见下段）。

3-8. There are four chains to the molecule, two light chains at the end that binds antigen (FAB [fragment that is antigen binding]), and two *heavy chains* running from the FAB end to the end that binds PMNL-receptors (Fc [fragment that is constant]). IgM and IgG activate (fix) complement; however, four types of IgG exist, called IgGl, IgG2, IgG3 and IgG4. IgG2 is poor at fixing complement and is therefore poor at bacterial killing and also a poor opsonin (see next paragraph).

免疫球蛋白的Fc片断部分在不同类型中结构是不同的，且具有不同的特征。有些抗体的Fc片断由被称为Fc-受体的宿主防御细胞受体所识别。吞噬细胞上最普通的受体是Fc γ-受体（Fc γ-R），它有几种亚型。Fc γ-R Ⅱ是PMNL-受体，被认为是结合IgG2调理细菌最重要的。Fc γ-R Ⅱ存在高和低亲合力的两种形式，1996年，Wilson和Kalmar发现患有侵袭性牙周炎的美国黑人的PMNL-受体具有低亲合力的Fc γ-R Ⅱ（即没有很好地结合IgG2）。这一重要发现（见第4章）并没有在患有侵袭性牙周炎的欧洲患者身上发现。

The Fc parts of Igs differ in structure between classes and have different properties. The Fc of some antibodies is recognised by host defence cell receptors called Fc-receptors. The most common receptor on phagocytes is the Fcy-receptor (Fcy-R) of which there are several subtypes. Fcy-RII is the PMNL-receptor thought to be most important for binding IgG2-op-sonised bacteria. There are high and low avidity forms of Fcy-RII, and in 1996, Wilson and Kalmar found that PMNL-receptors in black Americans who suffered from aggressive periodontitis had the form of Fcy-RII that was of low avidity (i.e. did not bind IgG2 very well). This key discovery (see Chapter 4) has not been found in European patients suffering from aggressive periodontitis.

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T 淋巴细胞

图 3-9 描述了基本的 T 细胞和 B 细胞免疫过程。可以看到 IL-1 和 IL-2 等细胞因子在此过程中起着主要作用。随着 T 细胞受体（未分化的 T 细胞表达 IL-2 受体）表达的，经抗原表达细胞（APCs）处理的抗原，与辅助 T 细胞（TH-1 cells）产生的 IL-2 结合，最终分化成为效应 T 细胞。

T-Lymphocytes

The basic T-cell and B-cell immune processes are represented in Fig 3-9. It can be seen that cytokines like IL-1 and IL-2 play a major role in this process. Following presentation of processed antigen by APCs to the T-cell receptor, that un-differentiated T-cell expresses IL-2 receptor, which binds to IL-2 produced by T-helper cells (TH-1 cells) and causes terminal differentiation into effector T-cells.

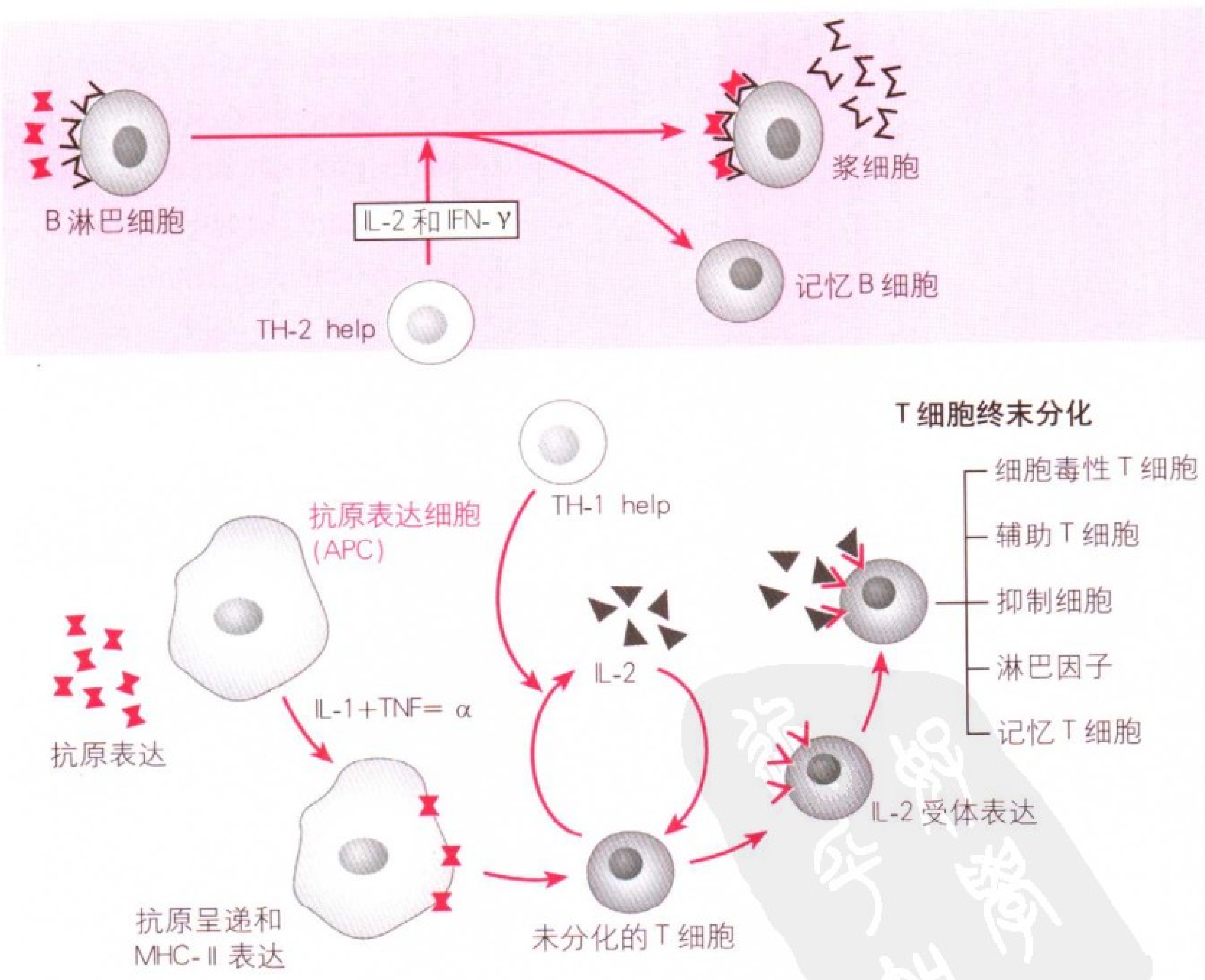


图 3-9　细胞介导和体液免疫反应

Fig 3-9 The cell-mediated and humoral immune responses.

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T细胞与B淋巴细胞在许多方面都不同（表3-3）。T细胞似乎是第一个到达龈炎损害部位。巨噬细胞和郎格汉斯细胞抗原表达细胞携带抗原到局部淋巴结，呈递给未分化的T细胞，T细胞开始转化，并经导航信号进入组织。导航信号仍是未知的，但细胞毒性T细胞到达组织后会以一种特殊的方式溶解细菌。记忆T淋巴细胞留在淋巴结内，发信号分子给产生淋巴因子的T细胞吸引巨噬细胞，防止它们从感染部位移走，它还行使其他许多功能，包括辅助B细胞功能［Ⅱ型辅助T细胞——(TH-2cells)］。Ⅰ型辅助T细胞（TH-1）辅助T细胞功能。

T-lymphocytes differ from B-lymphocytes in a number of ways (see Table 3-3). The T-cells seem to be the first to arrive in the gingivitis lesion. Macrophages and Langerhans cells (APCs) carry antigen to the regional lymph nodes, and present this to undifferentiated T-cells. The T-cells commence transformation and homing signals initiate their trafficking back to the tissues. The homing signals are not yet understood, but cytotoxic T-cells reach the tissues and lyse bacteria in a specific manner. Memory T-cells remain within the lymph nodes and signalling molecules (lym-phokines) from lymphokine producing T-cells attract macrophages, prevent their movement away from the infection and perform a variety of other functions including the assis-

表3-3　T细胞与B细胞的一些重要区别

Table 3-3 Some important differences between T-cells and B-cells.

特征	T细胞	B细胞
受体	TCR（T细胞受体复合体）	免疫球蛋白受体（IgD或IgM）
杀伤机制	T细胞介导与靶细胞接触	由游离免疫球蛋白介导和吞噬细胞介导（即B细胞未直接参与）
杀伤能力	T细胞活性需TH-1辅助	B细胞活性需TH-2辅助
亚型	种类多 细胞毒性T细胞 抑制T细胞 辅助T细胞 淋巴因子产生细胞 记忆细胞	种类少 浆细胞（活性免疫球蛋白分泌分泌管） 记忆细胞

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tance ofB-cell function [T-helper type 2 cells — (TH-2 cells)]. T-helper type 1 cells (TH-1) assist in T-cell functions.

B 淋巴细胞

B 淋巴细胞多数需要 TH-2 细胞帮助来产生抗体。有的B细胞活性不依赖 TH-2 的帮助，而是通过 T 细胞独立抗原。内毒素是一个重要的T细胞独立抗原的例子，在没有 TH-2 帮助下，具有多克隆B细胞活性。B细胞表面的免疫球蛋白受体与外来抗原的结合大多发生在局部淋巴结，但也可发生在组织内。当受到刺激时，B细胞增殖进而离开淋巴结，再次导回组织。以这种方式，一个受刺激的B细胞能产生大量克隆细胞来帮助宿主防御。当进入组织后，由于活跃的蛋白质合成作用，它们扩大形成浆细胞并产生免疫球蛋白，与抗原结合。如果浆细胞是在淋巴结内形成，则免疫球蛋白释放进入血液。抗原抗体复合物结合 PMNLs 上的 Fc 受体（调理素作用），通过经典途径激活补体。Ig- 细菌复合物的Fc 片断部分也结合C3b抗体再次调理PMNLs。浆细胞过大所以不能离开淋巴结只能释放抗体进入血液，经由血液到达组织。当免疫反应产生并确认为是活跃性牙周炎病损的特征时，浆细胞能在组织内形成，但出现在牙周炎的后期。非活跃性牙周炎病损是以T淋巴细胞为特征，伴有少量浆细胞。

B-Lymphocytes

B-lymphocytes largely require TH-2 cell help to produce antibody. Some B-cell activities can occur independently of TH-2 assistance, via T-cell independent antigens. Importantly, endotoxin is an example of a T-cell independent antigen, capable of polyclonal B-cell activation, in the absence of TH-2 help. Binding of foreign antigen by immunoglobulin receptors on the B-cell surface largely occurs in the regional lymph nodes, but may also occur in the tissues. When stimulated, B-cells proliferate and then leave the lymph nodes and again “home” towards the tissues. In this manner, one stimulated B-cell can produce large numbers of clones to assist in host defence. When in the tissues, they enlarge due to the active protein synthesis occurring within to form plasma cells and produce immunoglobulin, which is released to bind to antigen. If plasma cell formation occurs in the lymph nodes, then immunoglobulin is released into the blood stream. The antigen-antibody complexes bind Fc receptors on PMNLs (opsonisation) and activate complement via the classical pathway. The Fc part of the Ig-bacterial complex also binds C3b, which again opsonises PMNLs.

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Plasma cells are too large to leave the lymph nodes and release antibody into the blood stream, from where it reaches the tissues. Plasma cells can form within the tissues, but appear at the late stages of periodontitis, when the immune response is well developed and are believed to be characteristic of an *active* periodontitis lesion. The *inactive* periodontitis lesion is characterised by T-lymphocytes, with fewer plasma-cells.

临床结论

- 临床健康状态，PMNLs是主要的防御细胞，先天免疫功能在一个低水平状态，不会引起大量的组织破坏。
- 当牙菌斑形成时，内毒素和上皮毒素以及其他因素刺激补体激活，导致更大的炎症反应和牙龈组织的破坏。
- 炎症病损随时间变化而变化，T淋巴细胞出现，发生特异的可控制的细菌细胞杀伤。
- B淋巴细胞是确立病损的一个特征，能自由地产生抗体，与抗原结合并激活传统补体级联。
- 如对疾病易感患者不进行治疗，炎症反应的大小似乎会超过阈

Conclusions of Clinical Importance

- In clinical health, PMNLs are the predominant defence cell, and innate immunity functions at a low level, without causing significant tissue damage.
- As plaque builds up, the actions of endotoxin, epitheliotoxin and others stimulate complement activation, which leads to a greater inflammatory response and gingival tissue damage.
- With time, the inflammatory lesion changes and T-lymphocytes appear, providing specific and controlled killing of bacterial cells.
- B-lymphocytes characterise an established lesion and freely produce antibody, which binds to antigen and activates the classical complement cascade.
- If left untreated in disease-susceptible patients, the size of the inflammatory

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值，宿主组织破坏将以防御反应副作用的方式出现。

response seems to exceed a threshold where host tissue damage occurs as a side-effect of the defence strategy.

- 活跃的牙周病损由浆细胞支配，大部分组织破坏是炎症细胞释放酶和氧自由基，以"间接的"破坏方式完成的。

- Active periodontal lesions are dominated by plasma cells, and the bulk of the tissue damage arises as "collateral" damage following release of enzymes and oxygen radicals by inflammatory cells.

- 当酶和氧自由基水平超过宿主酶抑制剂和抗氧化剂的防御，则发生结缔组织破坏和骨吸收。

- When the levels of enzymes and oxygen radicals exceed the host's enzyme-inhibitor and antioxidant defence strategies, connective tissue damage and bone loss occur.

- 用抗炎药治疗牙周炎，也许能够调节免疫－炎症功能，但抗炎药不能在牙周组织达到有效的水平。所以治疗牙周炎的关键仍然是机械清除致病细菌。

- It is possible to modulate immune-inflammatory function in the treatment of periodontitis using anti-inflammatory drugs, but these do not reach into the tissues at significant levels. The key to therapy, therefore, remains the mechanical removal of the causative bacteria.

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第4章 危 险 评 估

Risk Assessment

目 的

危险评估已经成为疾病进程和随后治疗评估的基本方法。本章的目的是解释什么是危险因素以及它们与危险标志和危险患者的区别。将讨论牙周炎最重要的危险因素，并用一个简单的方案进行记录，这将使临床工作者在进行诊断、制定治疗计划和维持治疗时能考虑牙周病患者的危险因素在牙周病中的作用。

Aim

Risk assessment has become a fundamental strategy employed when assessing any disease process and its subsequent management. This chapter aims to explain what *risk factors* are and how they differ from *risk markers* and *risk patients*. The most important risk factors for periodontitis will be discussed and a simple protocol outlined that will enable practitioners to account for risk factors in their periodontal patients, when formulating diagnoses, treatment plans and maintenance regimes.

要 点

读完本章后读者将能正确评价危险因素分析在牙周评估中的重要作用，以及如何利用这些信息进行诊断，制定治疗计划和进行维持治疗。希望读者能够理解牙周病主要的危险因素，并能够在他们日常工作中通过一个简单快捷的评估系统进行评估。

Outcome

Having read this chapter the reader will appreciate the importance of the role of *risk factor analysis* in periodontal assessment and how such information informs diagnostic, treatment planning and maintenance strategies. It is hoped that the reader will also understand the key risk factors for periodontal disease and be able to implement a simple, rapid system of assessment

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within their daily practice.

危险评估

风险管理是一个在许多工作环境中都用到的术语，用来描述一系列实践中建立的常规安全方案。例如，从等候区到外科手术室入口的一级台阶可能增加患者进入手术室时被绊倒的危险。风险管理包括临床医师在工作中通过一个正式的危险评估过程来识别危险，确诊病症并引起患者的注意。如果失败将会对患者造成伤害，并且患者对治疗会产生抵制。在牙周病学中情况是相同的，由于不能识别已证实的牙周炎危险因素，使我们无法把患者口中存在的基本疾病问题告知并指导患者。而且，如果有的危险因素能被患者改变或消除，例如吸烟习惯或糖尿病的控制水平，医师就有责任恰当地告知患者他们自己在牙周病治疗中的作用。牙周炎的治疗要求牙科医师和患者之间的相互合作，成功的治疗均等地依赖患者的"一点"努力和牙科医生高水平的工作。

Risk Assessment

Risk management is a term used in many working environments to describe the formalisation of a series of safety protocols that put common sense into practice. For example, the presence of a step at the threshold of a doorway leading from the waiting area to the surgery is likely to increase the risk of a patient tripping as they enter the surgery. Risk management would involve the practitioner identifying the risk during a formal risk assessment for their practice, and erecting a sign to draw patients' attention to it. Failure to do so could result in injury to a patient and a claim against the practice. The situation in periodontology is similar, since failure to identify proven risk factors for periodontitis results in failure to inform and educate the patient in fundamental issues relating to the disease process in their mouths. Furthermore, given that some risk factors can be modified or eliminated by the patient, e.g. smoking habit or level of diabetic control, practitioners have a duty of care to counsel patients appropriately about their role in the management of their periodontal disease. Management of periodontitis is a contract between the dental surgeon and the patient in which the successful outcome of therapy is dependent equally upon the patient do-

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ing "their bit" as it is on the dental team performing their duties to the highest possible standard.

危险因素

危险因素是指增加个体疾病发生的可能性的因素。在生物学上危险因素与疾病的发生相关，但它们并不是必然原因与结果：即是说患者有某种危险因素并不意味他们一定会发生这种疾病。同样，没有某种危险因素也不意味疾病就不会发生。大多数科学家对疾病发生提出一个两级模型，在模型中主体（受实验者）必须具有对一种疾病的遗传危险因素，并获得环境危险因素，其能够开拓个体对疾病的固有的易感性。在牙周炎，危险因素被认为能改变基本致病过程（图4-1），并直接导致患病的可能性增加。它们可概括性地分为：

1. 系统危险因素（基于个体）——影响个体"系统"中能影响针对菌斑生物膜的宿主反应，打乱宿主－微生物平衡的一个系统的因素（图4-2）。

Risk Factors

A risk factor is a factor that increases the probability that a disease may develop in an individual. Risk factors are biologically related to the occurrence of the disease, but they do not necessarily imply cause and effect: i.e. just because a patient possesses a risk factor does not mean that they will definitely develop the disease. Equally, absence of a risk factor does not mean that the disease will not develop. The majority of scientists advocate a two-stage model for disease development, in which a subject must possess genetic risk factors for a disease and then acquire environmental risk factors capable of exploiting that individual's inherent susceptibility to the disease. In periodontitis, risk factors are believed to modify the basic pathogenic process (Fig 4-1) and such risk factors result in a direct increase in the probability of acquiring the disease. They may be broadly categorised as:

1. *Systemic risk factors* (or "subject-based") –factors affecting one of the subject's "systems", which essentially affect the host-response to the plaque biofilm, upsetting the host-microbial balance (Fig 4-2).

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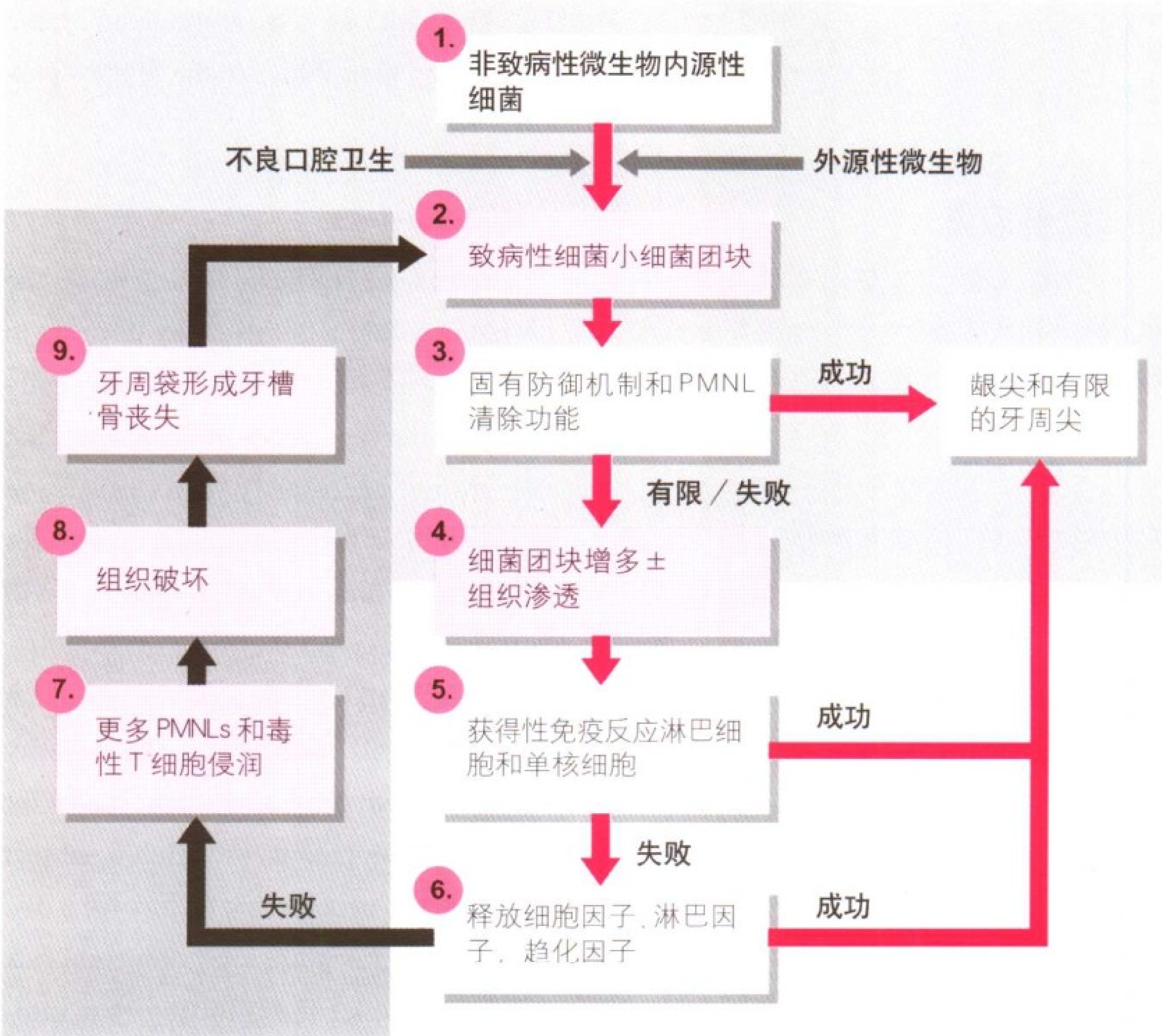


图 4-1 牙周病致病“关键途径模型”（引自 Salvi *et al.* 1997）

Fig 4-1 The “critical pathway model” for periodontal disease pathogenesis modified from Salvi et al. 1997.

2. 局部危险因素（基于位点）——口腔中的局部因素，可影响菌斑聚集或殆力。

危险标志

危险标志或预告因素与“因果关系”没有联系，但它们是疾病呈现的一个结果。危险标志暗示疾病的存在，在

2. *Local risk factors* (or “site-based”) – factors local to the oral cavity, which may influence plaque accumulation or occlusal forces.

Risk Markers

Risk markers or predictors are not linked to “causation”, but their presence is more a consequence of the disease being present.

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口腔临床体征变得明显前常被用来发现早期疾病。例如探诊出血（BOP）、牙齿松动、牙龈退缩、化脓，以前的疾病体验等（见第6章和第8章）。

Risk markers imply the presence of disease and are often used to detect early stages of disease, before overt clinical signs become apparent. Examples would be the sign of bleeding on probing (BOP), tooth mobility, recession, suppuration, past disease experience, etc. (see Chapters 6 and 8).

危险患者

危险患者是指在经过一个特定的周期后具有发生一种疾病的高可能性的患者。这类患者具有该疾病的危险因素或者暴露于该疾病的危险因素中。每天吸烟30支且口腔卫生不良的患者被认为是发生牙周炎或坏死溃疡性龈炎（NUG）的危险患者（见第6章）。

Risk Patients

A risk patient is one who has a high probability of developing a disease over a specific period of time. Such patients either possess or have been exposed to "risk factors" for that disease. Therefore, a patient who smokes 30 cigarettes per day and has poor oral hygiene must be regarded as a risk patient for the development of periodontitis or necrotising ulcerative gingivitis (NUG) (see Chapter 6).

为了帮助理解危险因素的作用，Offenbacher 在1996年（图4-1）建立了名为"致病关键途径模型"。该模型包含引起牙周破坏的细菌和宿主成分（图2-1），确定了疾病发生和进展时必须具备的九个关键阶段。每个闭合环周期代表进一步的组织破坏，危险因素可导致疾病在闭合环中从一个阶段到下一个阶段，或到另外的周期。同样，任何关键阶段可由于成功的宿主反应或由于危险因素的消除而逆转。

To help understand the role of risk factors, a model of disease pathogenesis that has been termed the "critical pathway model of pathogenesis" was developed by Offenbacher in 1996 (Fig 4-1). The model encompasses both the microbial and host components that contribute to periodontal destruction (Fig 2-1), but identifies nine key or critical stages in the process which must occur for disease development and progression. Each cycle of the closed loop represents further tissue destruction and risk factors may either lead to progress from one stage to the next within the loop, or to ad-

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ditional turns of the cycle. Equally, any critical stage may be reversed by successful host responses, or by elimination of risk factors.

多级危险评估

牙周疾病中关于危险评估的一个问题是牙周病是一个多因素疾病，致病菌单独出现并不足以导致疾病，所以牙周病的评估应在多级水平上进行。简言之，需要考虑四个水平级：

1. 患者水平　初次检查时进行。
2. 全口水平　初次检查和初次治疗后进行。
3. 牙齿水平　初次／最后治疗后和维持治疗后进行。
4. 位点水平　最后治疗后和维持期进行。

这种方法允许临床医师从牙周病进展或初次治疗失败的因素中分离可能引起牙周疾病的危险因素。图4-2描述了以上四个水平危险因素评估的实用性概括。

Multi-Level Risk Assessment

One of the problems with risk assessment in periodontal disease is that the diseases are multifactorial and assessment should therefore be at multiple levels. The presence of pathogenic bacteria alone is not sufficient to cause the disease. In simple terms, there are four levels to consider:

1. The patient level　Perform at initial examination.
2. The whole mouth level　Perform at initial examination and post-initial therapy.
3. The tooth level　Perform post initial/definitive therapy and maintenance.
4. The site level　Perform post definitive therapy and during maintenance.

This approach also allows the clinician to separate risk factors that may initiate periodontal disease from those responsible for its progression or for the failure of initial therapy. A practical view to the application of the above is illustrated in Fig 4-2.

患者水平危险评估

患者水平危险评估由第一次就诊时完成的以下内容来决定：

Patient-level risk assessment

Patient-level risk assessment can be determined at the initial consultation by performing the following:

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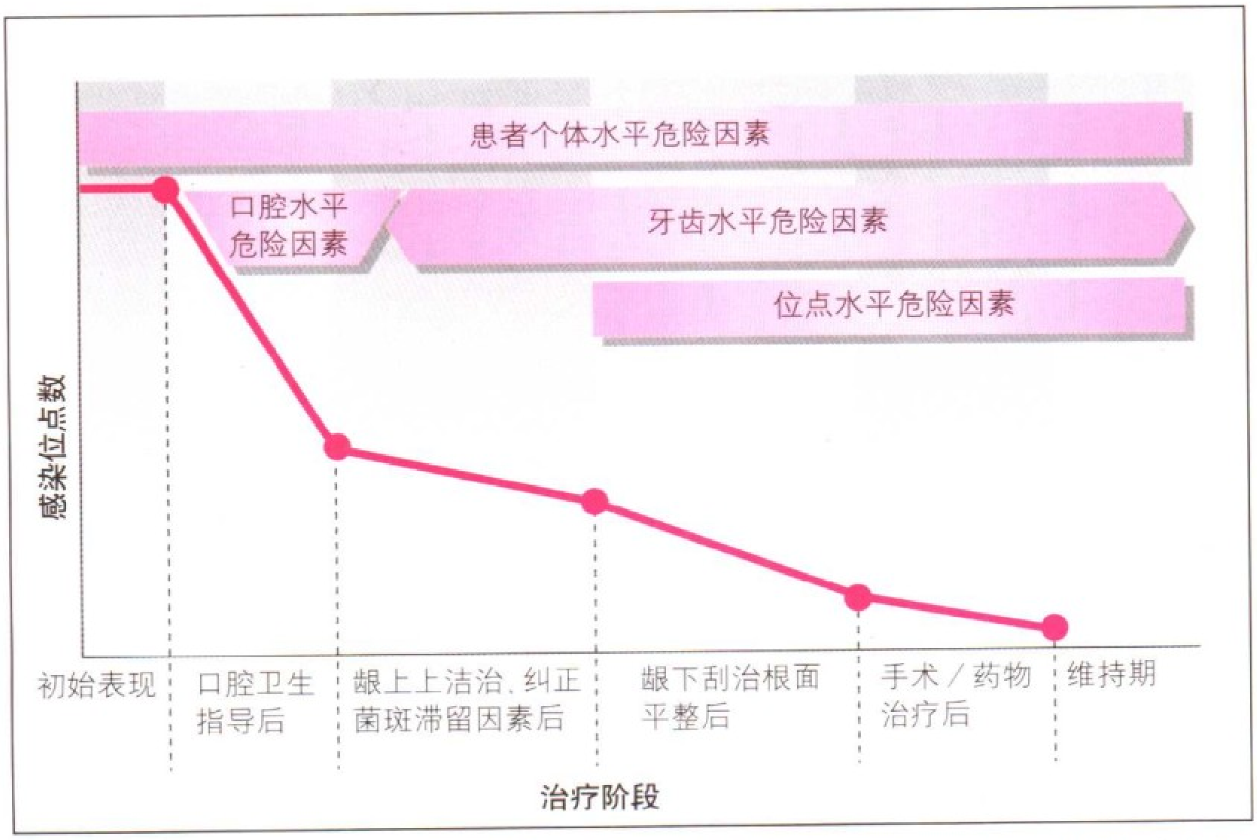


图 4-2　不同牙周治疗阶段的潜在效力和适用于每个治疗阶段的多个危险因素评估水平

Fig 4-2　The potential effectiveness of different stages of periodontal therapy, and how levels of multiple risk assessment can apply to each stage.

- 家族史（遗传性／先天性／遗传危险因素）。"家族中——父母／祖父母有牙龈疾病或牙槽溢脓或牙齿过早脱落吗？"
- 全身病史（系统性疾病，如糖尿病）。
- 牙科现病史（评价口腔卫生促动因素等）。
- 社会史（吸烟——现在或以往吸烟者）。
- 习惯（磨牙症）。

大多数医师常规收集这些信息，但建议尽可能进行与诊断同步的正规

- Family history (for hereditary/inborn/genetic risk factors). "*Is there a history of gum disease or pyorrhoea in the family – parents/grandparents – or early tooth loss?*"
- Medical history (for systemic diseases, e.g. diabetes).
- Present dental history (assess motivation to oral hygiene, etc.).
- Social history (smoking – current or former smoker).
- Habits (bruxism).

The majority of practitioners routinely collect such information, but its formal re-

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的记录。例如，使用带有四个系统性危险因素的记号盒子系统使该过程正规化（图 4-3）。

cording is recommended, perhaps alongside the diagnosis. For example, the use of a tick-box system with four key systemic risk factors would formalise this process (Fig 4-3).

全口水平危险评估

口腔水平危险评估在初次检查时，在对疾病易感性的基本牙周检查（BPE）后进行，像英国牙周病学界所推荐的那样（第 7 章），包括：

- 与年龄相关的附着丧失的检查。这是疾病先前／现在的进展速度的一个很好的概况，进而得知将来可能的进展速度。除了一个低的年龄相关的疾病经验，重要的是记住可能仍然发生局部病

Mouth-level risk assessment

Mouth-level risk assessment would be performed at the initial examination, after a Basic Periodontal Examination (BPE) for susceptibility – as recommended by the British Society of Periodontology (Chapter 7) – and would include:

- Examination of attachment loss relative to age. This gives a very good idea of previous/current rates of progression and thus the likelihood of future rates of progression. It is important to remember that rapid progression of

姓　　名：
编　　号：
出生日期：
诊　　断：慢性中度牙周炎
（UL67，UR5，LL21 和 LR12 重度病损）

系统危险因素：

家族史	☐	糖尿病	☐
吸烟者（包／年）	______	情绪紧张	☐
其他	______________		

图 4-3　简单记录卡

Fig 4-3 A simple tick-box system, which can be incorporated into an ink stamp and stamped in a susceptible patient's notes formally to record risk factors.

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损的快速进展。

• 静止状态的殆检查（殆类型，牙齿磨耗等）。2类Ⅱ分类错殆患者内倾的下切牙能导致这些牙齿出现舌面口腔卫生问题。

• 运动状态的殆检查（引导方式）。继发性殆创伤是对已有牙周疾病的牙齿的创伤，它能加快疾病进程，但它不会引发疾病（图4-4）。

local lesions may still occur, despite a low age-related disease experience.

• Occlusal examination in static relationship (occlusal class, tooth wear, etc). A class 2 division II malocclusion with retroclined lower incisors can create hygiene problems for lingual surfaces of those teeth.

• Occlusal examination in dynamic relationship (patterns of guidance). Secondary occlusal trauma is trauma to a tooth that is already periodontally diseased, and can accelerate the disease process, but does not initiate it (Fig 4-4).



a



b

图4-4　a. 继发性殆创伤牙，显示弧形骨吸收，此时必须首先治疗牙周炎；b. 原发性殆创伤牙，显示牙周膜间隙增宽和早期“漏斗状骨吸收”，创伤消除后可恢复正常

Fig 4-4 (a) A tooth subject to secondary occlusal trauma, as demonstrated by “crescentic” pattern of bone loss. In this situation, treatment of the periodontitis must take priority. (b) A tooth subject to primary occlusal trauma demonstrates widening of the periodontal ligament space and early “funnelling”. Elimination of the trauma will result in total bony resolution.

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- 口腔卫生水平的检查（图 4-5）。有时口腔卫生良好的牙周炎患者个体比口腔卫生差的牙周炎患者个体更有病情进展的危险。

- 菌斑滞留因素水平的检查（大量修复体存在，修复体的质量，牙石水平）。

- 活动性修复体的存在。一个设计良好的桥比一个可摘局部义齿的菌斑滞留少且更能让人满意（图 4-6）。

- 牙龈退缩水平。退缩程度越高，需要清洁的牙面就越大，滞留菌斑的危险就越大（图 4-7）。

- 牙龈组织的质地，口腔中与菌斑水平相关的牙周袋的范围（图4-8）。有的患者对牙周炎有抵抗力。

- Examination of levels of oral hygiene (Fig 4-5). A subject with periodontitis but excellent oral hygiene represents a greater risk of further disease progression than a subject with periodontitis and poor oral hygiene.

- Examination of levels of plaque-retentive factors (heavily restored mouth, quality of restorations, levels of calculus).

- Presence of removable prosthesis. A well-designed bridge is less plaque retentive and more desirable than a removable partial denture (Fig 4-6).

- Levels of recession. The greater the degree of recession, the greater the surface area to clean and hence the greater risk of leaving plaque (Fig 4-7).

- Gingival tissue quality and extent of pocketing relative to levels of plaque in the mouth (Fig 4-8). Some patients appear resistant to periodontitis.



图4-5 法医学上正规的卫生水平记录是重要的，但从患者出发记录存在/不存在是最基本的

Fig 4-5 Some formal recording of hygiene levels is important medico-legally, but from a patient motivation standpoint, disclosing is essential and dichotomous chart recording of "presence/absence" is essential.

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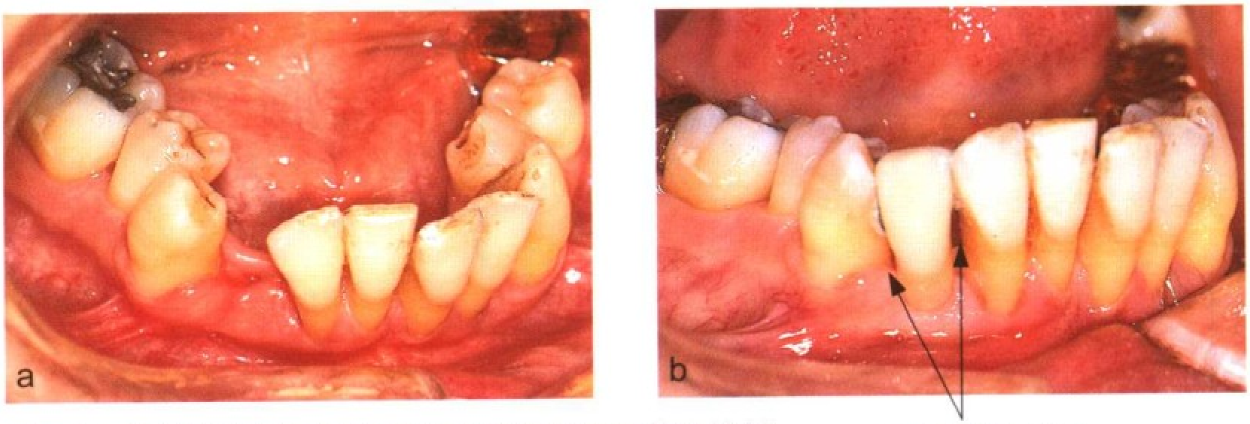


图 4-6　粘接桥有利于牙周炎患者良好的邻面菌斑控制，图中患者戒烟并进行全口洁治后成功的维持

Fig 4-6 An adhesive bridge designed to enable good interproximal plaque control in a periodontitis patient, currently on successful maintenance having quit a smoking habit and after full-mouth-open debridement.



图 4-7　无牙周袋无 BOP 患者，过大的刷牙力量导致附着丧失，UL6 需进行根管治疗

Fig 4-7 This subject has no pocketing or BOP, but excessive force during oral hygiene has resulted in attachment loss and a very large surface area to retain plaque free. The UL 6 has required root canal therapy.



图4-8　患者口腔卫生差，但无牙槽骨吸收和牙周袋，对牙周炎明显具有抵抗力

Fig 4-8 This subject demonstrates extreme oral neglect, but has no periodontal bone loss or pocketing. He/she is apparently resistant to periodontitis.

牙齿水平的危险评估

牙齿水平的危险评估可以在或不在初次检查时进行，一旦通过基础牙周检查发现了易感者，应完成详细的牙周检查图和详细的 X 线片评估和放射检查报告（第 8 章和第 9 章）。这部

Tooth-level risk assessment

Tooth-level risk assessment may or may not be carried out at the initial examination, once a BPE screen had identified a susceptible patient. In the latter situation, detailed periodontal charts and a detailed radio-

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分评估包括：

- 单个牙齿动度（松动度指数）。过度的松动可能有损患者功能或易使牙齿出现半脱位。
- 牙齿移位或漂移。牙周受损的牙齿在功能异常时可能移位，临床上经常看见上颌前牙出现类似情况，也是一种常见的主诉。
- 剩余牙齿支持（X线片）。X线检查剩余骨支持的范围能帮助决定长期的预后。骨组织支持少的慢性感染牙齿最好拔除，以减少潜在的病原体的集聚，它可能使治愈的和稳定的部位发生再感染。
- 根分叉病变的发生、位置和程度。根分叉部位（图4-9）更难维持，应检查牙齿活力。
- 个别牙的解剖［例如指状牙尖或鳞茎状牙冠（图4-10）］。
- 牙楔状隙和触点解剖（食物嵌塞、清洁能力）。
- 修复体台阶或修复体缺陷——在龈缘和龈缘下菌斑滞留（图4-11）。

graphic assessment and radiological report should be performed (Chapters 8 and 9). Part of this assessment would include:

- Individual tooth mobility (mobility index). Excessive mobility may compromise patient function or make a tooth susceptible to sub-luxation.
- Tooth movement or drifting. Periodontally compromised teeth may move under parafunction, a situation often seen with maxillary anterior teeth and a common presenting complaint.
- Residual tooth support (radiographically). The extent of residual radiographic bone support helps determine long-term prognosis. Chronically infected teeth with minimal bone support are best extracted to reduce the potential pathogen reservoir, which may re-infect treated and stable sites.
- Presence, location and extent of furcation lesions. Furcations (Fig 4-9) are harder to maintain and teeth should be vitality tested.
- Individual tooth anatomy [e.g. presence of "talon cusps" or bulbous crowns (Fig 4-10)].
- Anatomy of tooth embrasures and contact points (food packing, cleansability).
- Presence of ledges or deficiencies on restorations – retain plaque at or below the gingival margin (Fig 4-11).

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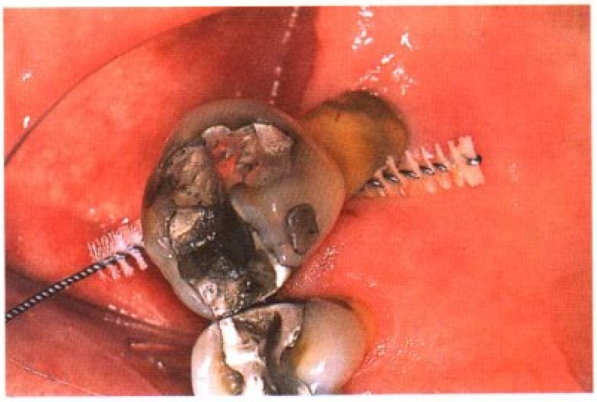


图4-9　根分叉病变需要特殊器械进行菌斑控制

Fig 4-9 Furcation lesions require special cleaning aids to maintain plaque free.



图 4-10　畸形牙尖导致龈缘菌斑滞留

Fig 4-10 Talon cusps retain plaque close to the gingival margin.



图 4-11　修复体悬突伴有牙槽骨吸收

Fig 4-11 A ledged restoration with associated bone loss.

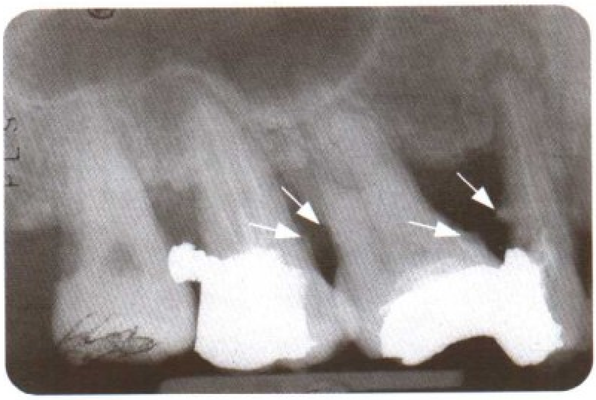


图 4-12　龈下牙石引起菌斑和毒素滞留，在易感患者引起局部骨缺损

Fig 4-12 Subgingival calculus, whilst inert, can retain plaque and toxins subgingivally, causing local bone defects in susceptible patients.

- 个别𬌗接触（早接触，过大的𬌗负荷，非工作侧接触等）。
- 软组织外形。
- 龈下牙石（图 4-12）。

上面所述中的一些，例如𬌗因素，或个别触点，可在治疗初期后进行评估，在那里口腔卫生措施和洁治已消除了大量的炎症区域，但是个别牙齿

- Individual occlusal contacts (prematurities, excessive loading, non-working-side contacts, etc.).
- Soft tissue contours.
- Subgingival calculus (Fig 4-12).

Some of the above, e.g. occlusal factors or individual contact points, may be assessed after the initial phase of therapy, where oral hygiene and scaling have re-

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仍对抗了初期治疗。

solved significant areas of inflammation, but individual teeth remain resistant to this initial therapy.

位点水平危险评估

位点水平的危险评估包括：

- 探诊出血。20%～30%的BOP位点是活动性的，而几乎全部经反复探诊无出血的位点则是非活动性的。但是吸烟状况可能影响这些统计数字。
- 溢脓。已治疗的牙周袋再溢脓暗示这些位点是不稳定的，要么需要再治疗，要么可能会发生根管感染。
- 局部牙根沟槽或牙根凹陷。对初次治疗没有反应的部位应该仔细探查寻找是否有沟槽或裂缝（图4-13）。
- 个别牙周袋探诊深度。这代表过去的疾病而非现在的活跃性，但治疗的目的是使牙周袋深度＜

Site-level risk assessment

Site-level risk assessment would include:

- Bleeding on probing. Approximately 20%–30% of BOP sites are active, whereas close to 100% of sites that repeatedly fail to BOP are inactive. However, smoking status may affect these statistics.
- Suppuration. Suppuration from a treated pocket indicates the site is unstable and either re-treatment is indicated, or an infected root canal may be present.
- Local root grooves or root concavities. Sites that fail to respond to initial therapy should be carefully explored for root grooves or cracks (Fig 4-13).
- Individual probing pocket depths. These represent historical disease rather than current activity, but the aim

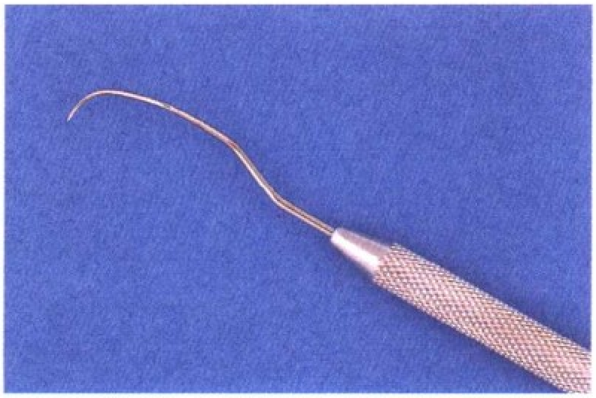


图4-13　ODU探针利于探查治疗无效的位点

Fig 4-13 An "Old Dominican university" (ODU) explorer is useful for fine exploration of non-responder sites.

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4 mm。

- 附着水平。
- 其他解剖因素［如釉珠，牙根沟槽（图 4-14）］。

位点水平的分析正常应在采取口腔卫生措施和洁治时，在那些特异部位的根面清创之前。在维持治疗初这些部位特征是重要的参考点，通过这些参考点来监控部位稳定性。

患者水平或系统危险因素

患者水平或系统危险因素主要分为：

A．遗传危险因素——例如当某些能产生毒素破坏 PMNLs 的牙周病原体存在时（第 2 章），PMNL 遗传的但较轻微的功能缺陷即可导致显著的功能异常。

of therapy is to reduce pocket depths to < 4mm.

- Attachment levels.
- Other anatomical factors [e.g. enamel pearls, root grooves (Fig 4-14)].

Site-level analysis would normally follow the hygiene and scaling phase of therapy and precede root-surface debridement of those specific sites that require it. The site-characteristics at the start of maintenance are the key reference point from which to monitor site stability.

Patient-Level or Systemic Risk Factors

Patient-level or systemic risk factors can be broadly categorised as:

A. Genetic/inherited/inborn risk factors – e.g. inherited but mild defects in PMNL function can lead to significant malfunction in the presence of certain periodontal pathogens (Chapter 2), which produce toxins to disable/destroy PMNLs.

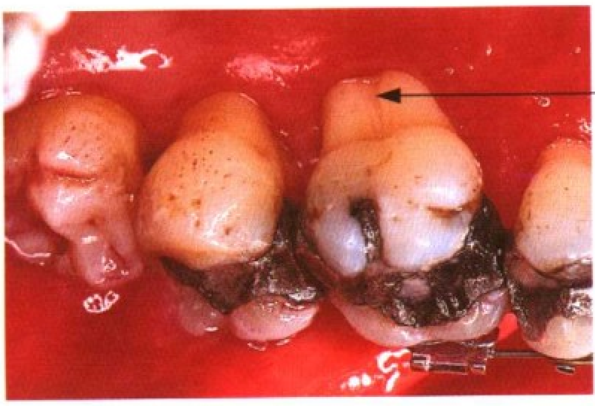


图 4-14　经反复非手术治疗失败后手术治疗暴露的腭根中央沟

Fig 4-14 A mid-palatal root groove, exposed during surgery after repeated attempts at a non-surgical approach to therapy failed.

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B. 环境危险因素——例如常用苯妥英类药物（如大仑丁）来治疗癫痫，钙通道阻断药（如氨氯地平）治疗高血压和心绞病，或服用环孢素防止移植手术后的器官排斥反应。

C. 行为危险因素——例如吸烟，现在被认为是牙周病非常重要的危险因素。

D. 生活方式危险因素——例如社会心理压力是坏死溃疡性龈炎的一个强烈的危险因素（第6章）。有证据表明情绪紧张也是慢性牙周炎一个潜在的危险因素。

E. 代谢危险因素——例如糖尿病1型和2型，能显著地提高患者对牙周炎的易感性。

F. 血液病学的危险因素——例如白细胞黏附缺陷（LADs）或白细胞惰性综合征。

这些危险因素可出现双重分类，例如糖尿病具有很强的遗传基础，但是糖尿病本身与高血糖相比可能不是牙周炎的危险因素，而高血糖状态将改变PMNL功能、结缔组织和脂肪代谢。

B. Environmental risk factors–e.g. drug therapies involving phenytoin (e.g. Dilantin®), used to treat epilepsy, calcium channel blockers (e.g. amlodipine), used to treat hypertension and angina, or ciclosporin, used posttransplantation to prevent organ rejection.

C. Behavioural risk factors – e.g. smoking, which is now recognised as a highly significant risk factor for periodontal disease.

D. Life-style risk factors – e.g. psychosocial stress, which is a strong risk factor for NUG (Chapter 6). Evidence is emerging that stress is also a potential risk factor for chronic periodontitis.

E. Metabolic risk factors – e.g. diabetes mellitus types I and II, which significantly enhance susceptibility to periodontitis.

F. Haematological risk factors – e.g. leucocyte adhesion deficiencies (LADs) or lazy leucocyte syndromes.

Certain risk factors may appear to be of dual classification. For example, diabetes has a strong genetic basis, but the diabetic state per se may not be the risk factor for periodontitis, rather the hyperglycaemic state, which alters PMNL function and connective tissue and lipid metabolism.

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牙周炎遗传危险因素

对双胞胎、兄弟（姐妹）和家族的研究证据表明在侵袭性牙周炎中存在相似的情形。在常染色体显性遗传中，这种遗传方式的基因与孟德尔式遗传的主要基因或相关基因一致。然而根本的危险因素大多尚未清楚、明白。而且，负责这种易感性的基因在人种和地区之间可能不同，因为PMNL缺陷在一个侵袭性牙周炎患者群中被发现，而在其他人群中却没有产生。不过侵袭性牙周炎的家族史有助于医师设计规律的复诊计划和提高复诊时检查的详细程度。对附着丧失和活动性牙周袋形成的早期诊断大大地简化了治疗。

近来，注意力都集中在了单核苷酸多态性（SNP）分析上。单核苷酸多态性是一个可变的基因结构部位，一个核苷酸不同其本质就不同。这会导致产生不同效果的蛋白质。该蛋白是PMNL-受体或炎症前细胞因子，对炎症-免疫反应的影响是巨大的，此蛋白水平升高在确定个体对牙周炎易感性

Genetic risk factors for periodontitis

There is good evidence from twin, sibling-pair and family studies that a familial pattern exists with certain forms of aggressive periodontitis. The pattern of transmission is consistent with Mendelian inheritance of a major gene or linked genes, in an autosomal dominant manner. However, the underlying risk factors remain largely obscure and are poorly understood. Furthermore, the genes responsible for such susceptibility may differ between races and continents, since PMNL defects found in one population of aggressive periodontitis patients have not been reproduced in others. Nevertheless, knowledge of a family history of aggressive periodontitis helps the practitioner to design regular recall programmes and to increase the level of detail of examinations performed at such recalls. Early diagnosis of attachment loss and active pocket formation greatly simplifies therapy.

More recently, attention has focused on single nucleotide polymorphism (SNP) analysis. SNPs are variable regions in a gene structure that essentially differ by one nucleotide only. This can result in the production of proteins of differing effectiveness. Where such a protein is a PMNL-receptor or pro-inflammatory cytokine, the

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方面可能有一定作用（见框4-1）。在人群中普遍存在的基因多态性未必能解释像牙周炎这种相对非普遍性疾病（在人群中占10%～15%），这似乎是符合逻辑的。所以测定现在已知的各种基因多态性的不同组合的临床效果的工作已经开始。表4-1概括了一些由于白细胞质或量的缺陷所引起的遗传疾病，而牙周炎是它们的一个特征。

effect on the inflammatory-immune response can be substantial and may play a role in determining the increased susceptibility of an individual to periodontitis (see Box 4-1). It seems logical that gene polymorphisms that are common in the population are unlikely to explain a disease like periodontitis, which is relatively uncommon (10–15% of the population). Work has therefore begun to determine the clinical effects of different combinations of the vari-

表4-1 影响牙周组织的遗传性疾病

Table 4-1 Inherited conditions affecting the periodontal tissues.

疾 病	牙周相关根本缺陷
Down 综合征	PMNL 趋化性、杀伤和吞噬功能缺陷。T细胞抗原诱导杀伤作用降低
慢性肉芽肿性疾病	吞噬细胞“呼吸爆发”失败。氧自由基没有产生，细菌得以生存
胰岛素依赖性青少年糖尿病	高血糖状态降低了PMNL功能。单核细胞处于高反应状态，产生过多的IL-1β，PGE_2，TNF_2和氧自由基。对胶原溶解性和血管分布的影响延缓愈合
低磷酸酯酶血症	低水平的碱性磷酸酶导致牙骨质矿化/形成不良和牙齿片状剥落
掌跖角化-牙周破坏综合征	PMNL趋化性和吞噬作用异常。PMNL酶（组织蛋白酶-C）基因在基因图谱中位于染色体11
先天性结缔组织发育不良综合征	胶原合成异常——Ⅷ型与重度牙周组织破坏有关
先天性白细胞颗粒异常综合征	吞噬细胞趋化性、脱（颗）粒和细胞膜融合异常。全口牙脱落
Jobs 综合征	肥大细胞释放过多IgE和组胺以及IgE免疫复合体形成

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ous gene polymorphisms that are currently known about. Table 4-1 summarises some genetically inherited disorders, where periodontitis is a feature, due to qualitative or quantitative defects in leucocytes.

框 4–1

牙周炎中被调查的单核苷酸多态性例子

1996 年，Wilson 和 Kalmar 检测了与 IgG2 结合的 PMNL 受体的多形性，即所谓的 FcgR Ⅱ a（Fc-gamma receptor Ⅱ a）。存在两种同种异型，一个高亲和力受体（H131—在染色体 131 位编码精氨酸）和一个低亲和力受体（R131—在 131 位编码组氨酸）。他们发现患有局部早发性（侵袭性）牙周炎的黑人患者具有低亲和力受体的频率增加，导致 PMNLs 与 IgG2 结合的能力减弱。Kornman 等在 1997 年调查了 IL-1 基因位点的单核苷酸多态性并证明牙周炎与特异的过多产生 IL-1 的有关基因型［单倍体 IL—1α $^{-889}$（等位基因 2）和 IL-1β $^{+3953}$（等位基因 2）］有关。他们称之为 PST（+）基因型，并证明在一个小的志愿者群中它与慢性牙周炎易感性增加相关。现在通过椅旁分析检测指血可检测到。然而，这个基因多态性只对吸烟的患者具有预示性，当应用于大量受

Box 4-1

Examples of SNPs Investigated in Periodontitis

In 1996, Wilson and Kalmar examined a polymorphism in the PMNL-receptor that binds IgG2, the so-called FcgRIIa (Fc-gamma receptor Ha). Two allotypes exist, a high-affinity receptor (H131 – arginine coded for at position 131 on chromosome) and a low-affinity receptor (R131 – his-tidine coded for at position 131). They found that Black sufferers of localised early-onset (aggressive) periodontitis had an increased frequency of the low-affinity receptor, resulting in their PMNLs binding poorly to IgG2. Kornman et al. investigated a SNP in the IL-1 gene locus in 1997 and demonstrated that periodontitis was associated with a specific genotype (haplotype IL1- α $^{-889}$ [allele 2] with IL1- β $^{+3953}$ [allele 2]) that coded for excessive IL1 production. They called this the PST(+) genotype and showed that it was associated with in-

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试者时，其作为诊断工具的能力明显降低。在英国人群中进行的类似的研究不能证明这一关系。

creased susceptibility to chronic periodontitis in a small group of volunteers. A finger-prick blood test is now available for chair-side analysis. However, this polymorphism appeared predictive only for subjects who also smoked, and when applied to larger numbers of subjects, its power as a diagnostic tool reduced considerably. Similar studies in a UK population have failed to demonstrate such a relationship.

牙周炎环境危险因素

药物如：

- 苯妥英 （50%）
- 钙通道阻断药（如硝苯地平、氨氯地平、非洛地平）（5%～20%）
- 环孢素 （30%）

能导致药物性牙龈增生、假性牙周袋和随后真性牙周袋的形成。通常通过他们的内科医师可以将患者的抗高血压的首选药物改为非钙通道阻断药。在移植术患者中很少因为牙龈增生这个指征而使用替换药物（如他克莫司）替代环孢素；这可以通过与负责控制移植患者药物的内科医师会诊实现。

Environmental risk factors for periodontitis

Drugs such as:

- phenytoin (50%)
- calcium channel blockers (e.g. nifedipine, amlodipine, felodipine) (5%-20%)
- ciclosporin (30%)

can lead to drug-induced gingival overgrowth, false pocketing and subsequently true pocketing. It is often possible to change a patient's anti-hypertensive drug regime through their medical practitioner to a non-calcium channel blocker, which should be the first approach. One of the few indications for withdrawal of ciclosporin in transplant patients and substitution with an alternative (e.g. tacrolimus) is gingival overgrowth; again this can be achieved

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through consultation with the physician responsible for the transplant patient's medical control.

艾滋病

研究证明坏死溃疡性龈炎(NUG)，坏死溃疡性牙周炎（NUP）和坏死性口炎（NS）在HIV阳性患者中存在更加普遍（第6章），慢性牙周炎基线附着水平与血清阴性对照没有差异。另外，假如口腔卫生水平是可比较的，HIV感染者的附着丧失与非感染对照组相比，附着丧失进展速度并不是显著增高。

HIV disease

Research has demonstrated that whilst conditions such as necrotising ulcerative gingivitis (NUG), necrotising ulcerative periodontitis (NUP) and necrotising stomatitis (NS) are more common in HIV-positive patients (Chapter 6), baseline attachment levels in chronic periodontitis are no different to sero-negative controls. Additionally, attachment loss does not appear to progress at a significantly higher rate in HIV-infected over non-infected controls, provided oral hygiene is comparable.

牙周炎的行为危险因素

牙周疾病最重要的两种行为危险因素是：

- 不良的口腔卫生
- 吸烟

Behavioural risk factors for periodontitis

The two most important behavioural risk factors for periodontal diseases are:

- poor oral hygiene
- smoking.

不良的口腔卫生危险因素

口腔卫生问题是牙周疾病中最重要的因素。如图4-2所示，许多研究证明随口腔卫生的改善，牙龈炎症患者的龈袋探诊深度明显降低，牙周附着明显增加。患者有效清洁的能力和他们进行有效清洁的动机是独立的问题。聪明的患者明白，他们口腔卫生水平

Poor oral hygiene as a risk factor

The issue of oral hygiene is the most important factor in periodontal disease. As indicated in Fig 4-2, many studies have demonstrated significant reductions in probing pocket depths, attachment gains and, of course, in gingival inflammation, with improvements in oral hygiene alone.

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需要比90%的没有患牙周炎症危险的人群要高，他们有责任关心他们自己，而且只体现能在每天的日常生活中。有必要通过记录菌斑和出血指数来促进和监控口腔卫生水平，并反馈给患者，对于反复不能达到获得理想牙周健康所必需的口腔清洁水平的患者，告知其必须接受疾病会进展的可能性，而且更适合姑息治疗以尽可能延长牙列的保留时间。

The patient's ability to clean effectively and their motivation to do so are separate issues. However, dextrous patients who understand that their level of oral hygiene needs to be higher than that of the 90% of the population that are not at risk from periodontitis have a duty of care to themselves, which only they can fulfil on a daily basis. It is essential to motivate and monitor levels of oral hygiene by recording plaque and bleeding scores and to feed these back to the patient. The patient who repeatedly fails to achieve the levels of cleaning necessary for optimal periodontal health must accept the likelihood of disease progression and is more suited to a palliative treatment regime to maintain their dentition for as long as possible.

吸烟危险因素

现在相当多的证据支持吸烟是牙周病最重要的危险因素之一。

吸烟对牙周膜的影响是广泛的，吸烟者：

- 具有更多的深牙周袋位点
- 具有更多的临床牙周附着丧失/骨丧失水平
- 具有更普遍的根分叉病变
- 堆积更多牙石
- 证明吸烟习惯（pack years=每天吸烟盒数×吸烟年数）和牙周破

Smoking as a risk factor

A considerable body of evidence now supports smoking as one of the most significant risk factors for periodontal disease.

The effects of tobacco smoking on the periodontium are broad and smokers:

- have more sites with deeper pockets
- have greater levels of clinical attachment loss/bone loss
- have a higher prevalence of furcation lesions
- accumulate more calculus
- demonstrate a dose response between smoking habit (pack years = number

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坏之间存在剂量反应。

吸烟与牙周疾病之间的联系，首次建立于 1946 年，是由于吸烟和 NUG 的联系。具有讽刺意味的是，香烟中的尼古丁和其他成分引起血管收缩，减少牙龈出血和牙龈红色（也由于牙龈表面的角化），而掩饰了牙周炎症的重要临床体征。

吸烟患者是非吸烟患者牙周病水平的 3～6 倍（依标准），而 19～30 岁的吸烟患者患牙周病的可能性是非吸烟患者的四倍多。研究表明每天吸 1 支烟将增加 0.5% 的附着丧失，每天 10 支则增加 5%，每天 20 支增加 10%。90% 的难治性牙周炎病例（第 6 章）是吸烟者（普通人群为 25%）。

吸烟患者的手术治疗和非手术治疗效果均较差，吸烟患者在牙周维持治疗期失牙的可能性是非吸烟患者的两倍多。在手术后牙周组织得到改善的同时，吸烟患者比非吸烟患者经历：

- 较少牙周探诊深度减少
- 获得很少的临床牙周附着
- 获得较少的骨高度

吸烟作为一个危险因素的潜在机

of packs per day × number of years patient has smoked) and periodontal destruction.

The link with periodontal diseases was first established in 1946 owing to an association between smoking and NUG. Ironically, nicotine and other agents within smoke cause vasoconstriction, reducing gingival bleeding and gingival redness (also owing to surface keratinistion of gingivae), and hence mask important clinical signs of periodontal inflammation.

Smokers have three to six times (depending on criteria) the level of periodontal disease of non-smokers, and 19–30-year-olds are four times more likely to suffer. Studies have shown that one cigarette per day increases attachment loss by 0.5%, ten per day by 5% and 20 per day by 10%. Ninety per cent of refractory periodontitis cases (Chapter 6) are smokers (compared with 25% of general population).

Non-surgical therapy and surgical therapy are less effective in smokers, who are two times more likely to lose teeth during periodontal maintenance. Whilst the periodontal tissues do improve with therapy, smokers experience:

- less probing pocket depth reduction
- less clinical attachment gain
- less bone height gain.

The mechanisms underlying smoking as

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制非常复杂，但可能包括：

- 福赛类杆菌的流行↑
- 更难以根除 *Aa*，*Pg* 和 *Bf*
- 唾液 IgA 水平↓
- 早发性（侵袭性）牙周炎吸烟患者的血清 IgG_2↓
- 吞噬细胞溶酶体酶和氧自由基的释放↓
- T-辅助细胞：T-抑制细胞的比例↓
- LPS 刺激单核细胞 TNF2、IL1-β 和 IL-6 的释放↑
- 最初牙龈血液流速↓（长期吸烟者可补偿/自我纠正）
- 细胞基质蛋白的破坏↑
- 牙龈角化和纤维变性↑

无烟烟草（用鼻吸或咀嚼烟草）与咀嚼烟草相邻位置的附着丧失有关（主要在下颌磨牙的颊侧）。

戒烟改善患者对治疗的反应介于吸烟和不吸烟者中间，尽管完全获得这一好处需要1到10年不等。大多数患者每6个月就要见他们的牙医，而相对少地见他们的GP（全科医师），所以牙医们在劝告戒烟的作用变得很重要。吸烟习惯在妇女和年轻人中是一个主要问题，尤其这两个群体经常用到牙科服务。实际上为检测主要的尼古丁代谢物的2min椅旁唾液检测在我们的实验室和合作者中已开展起来。在普

a risk factor are complex, but may include:

- ↑ prevalence of *B.forsythus* (Bf)
- Harder to eradicate *Aa, Pg and Bf*
- ↓ levels of salivary IgA
- ↓ serum IgG2 in smokers with early-onset disease (aggressive)
- ↓ release of lysosomal enzymes and oxygen radicals by phagocytes
- ↓ ratio of T-helper: T-suppresser cells
- ↑ release of TNFα, IL1-β, IL-6 by monocytes when stimulated by LPS
- ↓ gingival blood flow initially (may compensate/self-correct in chronic smokers)
- ↑ destruction of extra-cellular matrix proteins
- ↑ gingival keratosis and fibrosis.

Smokeless tobaccos (snuff and chewing tobacco) are associated with attachment loss adjacent to the site of chewing (mainly buccal of mandibular molars).

Cessation of smoking improves response to therapy to mid-way between non-smokers and smokers, though figures vary from one to ten years for the full benefits to be realised. The role of the dental team in smoking cessation counselling is becoming high profile, as most patients visit their dental practitioner every six months, but visit their GP relatively rarely. Two groups - females and young adults - where a smoking habit remains a major problem, in

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通的牙科实践中进行的一项研究证实，当它被用作辅助普通牙医劝告戒烟时，六周戒烟比率有明显提高。

particular, regularly make use of dental services. Indeed, a two-minute, chair-side saliva test for major nicotine metabolites has been developed in our laboratories and those of collaborators. A study performed in general dental practice demonstrated significant improvements in six-week quit rates, when this was used as an adjunct to smoking cessation advice given by a general dental practitioner.

牙周炎的生活方式危险因素

Life-style risk factors for periodontitis

近年来，社会心理压力和较小程度上饮食和营养的作用在牙周病研究中受到关注。

The roles of psychosocial stress and to a lesser extent diet and nutrition have recently attracted attention in periodontal research.

紧张危险因素

大量研究调查了作为牙周病危险因素的情绪紧张、忧伤和模仿行为。因为经济紧张和抑郁症降低了牙科就诊率和口腔健康意识，很难取得联系，所以疾病水平增高可能是由于忽视了口腔健康而非生物学原因。然而NUG和情绪紧张之间的联系是确立的，情绪紧张确实降低了免疫功能。已证明情绪紧张降低了唾液流速，增加了唾液黏滞度、酸度和糖蛋白成分，进而有助于更大的菌斑形成。最近，已证明由于慢性情绪紧张导致升高的去甲肾上腺素和肾上腺素水平能减少牙龈血流。我们实验室的工作已证明确定的牙周致病菌能够利用龈沟液内的儿茶酚胺

Stress as a risk factor for periodontitis

A number of studies have investigated stress, distress and coping behaviours as risk factors for periodontal disease. Associations are difficult to make, because financial strain and depression reduce dental attendance rates and oral health awareness, hence increased levels of disease may be due to oral neglect rather than biological reasons. However, the link between NUG and stress is well established and stress does reduce immune function. Stress has also been shown to reduce saliva flow and to increase salivary viscosity, acidity and glycoprotein content, thus favouring greater plaque formation. More

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帮助它们进行增殖，帮助他们在敌对的、铁限制性的龈沟环境中幸存下来。情绪紧张仍然是牙周炎的一个潜在的危险因素。

recently, elevated levels of noradrenaline and adrenaline, due to chronic stress, have been shown to reduce gingival blood flow. Work in our own laboratories has shown that certain periodontal pathogens can utilise these catecholamines within GCF to help them multiply, aiding survival in the hostile, iron-restrictive environment of the gingival crevice. Stress remains a potential risk factor for periodontitis.

营养不良危险因素

很少有研究把注意力放在饮食对牙周病的致病作用上。已证实个别维生素缺乏（Vit C）引起牙龈出血，近年来的研究证明柑橘的摄入能改善PMNL的功能，但需要对一个平衡的饮食和抗氧化剂复合物的效果进行评估，而不是单独的一个。考虑到氧自由基在牙周组织破坏中的作用，抗氧化剂似乎是将来研究的一个好的目标。目前饮食只是牙周炎的一个潜在危险因素。

Malnutrition as a risk factor for periodontitis

Very little research has addressed the role of the diet in periodontal disease pathogenesis. Individual vitamins (vitamin C) have been shown to affect gingival bleeding and recent work has shown improved PMNL function with citrus fruit intake, but work is needed to assess the effects of a balanced diet and complexes of antioxidants, rather than individual ones. Given the role of oxygen radicals in periodontal tissue destruction, antioxidants would seem a good target for future research. At present, diet is only a potential risk factor for periodontitis.

牙周炎危险因素——代谢疾病／紊乱

这个名单包括：

- 糖尿病
- 妊娠和口服避孕药物

Metabolic diseases/disorders as risk factors for periodontitis

This list includes:

- diabetes mellitus
- pregnancy and the oral contraceptive

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- 骨质疏松症
- 克罗恩病
- 结节病

性激素对青春期和妊娠期牙龈炎症的影响已被清楚地认识。我们知道妊娠期性激素水平升高可导致更高水平的龈炎，但牙槽骨的加速吸收可能继发于牙龈炎症，或由于口腔卫生习惯改变所致。早期证据提示骨质疏松症与牙槽嵴高度降低之间有关系，但是这也可能是更一般性的骨质减少的一个表现。

- osteoporosis
- Crohn's disease
- sarcoidosis.

The effects of sex hormones during puberty and pregnancy on gingival inflammation are well recognised. Higher levels of gingivitis due to sex hormone increases during pregnancy are also recognised, but links to accelerated bone loss may be secondary to gingival inflammation, or due to behavioural changes in oral hygiene practices. Early evidence suggests a link between osteoporosis and alveolar crestal height loss, but again this may be a manifestation of a more generalised osteopenia.

糖尿病危险因素

当糖尿病控制不良时，1 型（胰岛素依赖型）和 2 型（非胰岛素依赖型，年龄发作，饮食控制）糖尿病患者增加了感染牙周炎和其他口腔感染的危险。尤其：

- 随年龄增加，青春后期牙周炎的发病率↑
- 糖尿病的全身并发症越严重，牙周炎就越严重

牙周炎与糖尿病的关系与菌斑控制和牙石无关。

糖尿病的长期控制（30～90 d 以上）是通过血糖水平的评价来衡量，因

Diabetes mellitus as a risk factor for periodontitis

Type I (formerly insulin-dependent) and type II (formerly non-insulin-dependent, age-onset, diet-controlled) diabetics are at increased risk for periodontitis and other oral infections, where diabetic control is poor. Specifically:

- Incidence of periodontitis post-puberty ↑ with age.
- The greater the systemic complications of diabetes the more severe the periodontitis.

The relationship with diabetes appears independent of plaque control or calculus.

Long-term diabetic control (over 30-90 days) is measured by assessing glycated

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为高血糖引起葡萄糖与血红蛋白(Hb)不可逆结合。两种血红蛋白中的一种可按以下标准进行评估：

- HbA1——正常检测水平<8%的血红蛋白。
- HbA1c——正常水平为血红蛋白的6.0%~6.5%。

1型糖尿病有以下特点：

- 随着年龄↑，发生牙周炎的危险↑。
- 随着糖尿病持续时间↑，疾病的严重程度↑。
- HbA1/HbA1c的水平越高，骨吸收越重，特别是在邻间位置。

2型糖尿病与牙周病的相关性研究已大量在皮玛印弟安人群中进行，皮玛人的2型糖尿病的发病率很高。研究结果显示：

- CAL和骨丧失是正常人的3倍。
- 患有糖尿病的年轻皮玛人中牙周疾病患者是正常人的5倍多。
- 年轻受试者具有4倍多疾病进展的可能。
- 血糖控制较差的地方，患牙周炎的危险增加10倍。

对糖尿病患者起作用的危险因素

haemoglobin (Hb) levels, since hyperglycaemia causes glucose irreversibly to bind to Hb. One of two types of Hb can be assessed:

- HbAl–normal test levels <8% of Hb.
- HbAl c – normal levels 6.0-6.5% of Hb.

Type-I diabetics have:

- ↑ risk of developing periodontal disease with ↑ age.
- Disease severity ↑ with ↑ duration of diabetic condition.
- The higher the HbAl/HBAlc level the worse the bone loss, especially at interproximal sites.

Type II diabetes has largely been studied in relation to periodontal disease in a population of Pima Indians where the incidence of type II disease is high. Results show:

- Three times greater likelihood of CAL and bone loss.
- Younger Pima Indians with diabetes had five times more periodontal disease.
- Younger subjects were four times more likely to experience disease progression.
- Where glycaemic control was poor, the risk was 11-fold higher for periodontitis.

Factors that appear to contribute to the

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包括：

- PMNL 功能——已证实存在受损的趋化作用、吞噬作用和黏附功能。PMNL 功能异常可能是遗传所致或可能继发于高血糖。
- 胶原代谢——糖尿病患者牙龈成纤维细胞合成胶原减少，PMNL 胶原蛋白酶水平↑。
- AGE 产物——在高血糖环境中，蛋白质结构经非酶糖化形成糖化末端（AGE）产物。AGE 产物导致胶原X键连接↑，进而↓溶解度／逆转。单核细胞／巨噬细胞也有AGE受体，当AGE产物与之结合时刺激释放IL-1，TNF α，PGE_2和氧自由基。
- 伤口愈合——由于胶原溶解度↓和胶原蛋白酶使新形成的胶原去除↑，糖尿病患者伤口愈合↓。

实用点

大多数 1 型糖尿病患者在接受牙科治疗前已被诊断明确，同时，2 型糖尿病患者仍然可能最先出现在口腔医师面前。图4-15a中的患者表现出一个非典型的外侧牙周脓肿，它对简单的

risk in diabetic patients include:

- PMNL function–impaired chemotaxis, phagocytosis and adherence have been demonstrated. The PMNL defects may be genetically inherited or may be secondary to the hyperglycaemia.
- Collagen metabolism – gingival fibroblasts from diabetic subjects synthesise less collagen and levels of PMNL collagenase are ↑.
- AGE products – in a hyperglycaemic environment protein structures undergo non-enzymatic glycation to form advanced glycation end (AGE) products. AGE products cause ↑ collagen x-linkage and thus ↓ solubility/turnover. Also, monocytes/macrophages have AGE-receptors, which when bound by AGE products stimulate release of IL-1, TNFα, PGE_2 and oxygen radicals.
- Wound healing – is ↓ in diabetics due to the ↓ collagen solubility and ↑ collagenase removal of newly formed collagen.

Practical points

Whilst most type I diabetics are diagnosed prior to dental attendance, type II diabetes can still present initially to the dental surgeon. The patient in Fig 4-15a presented with an atypical lateral periodon-

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冲洗和根面刮治治疗无反应。尿液分析条的使用（图4-15b）证实，尿糖水平高时，随机血糖测试（随机而不是禁食葡萄糖8h）证实显著高血糖。

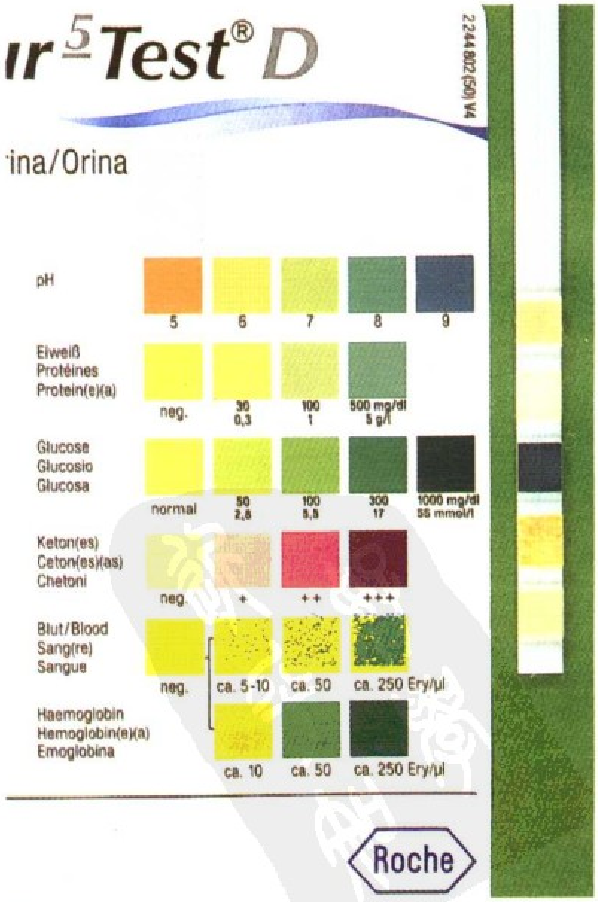
总之，糖尿病控制越好，对牙周治疗的反应越佳。控制良好的糖尿病患者可同任何其他患者一样进行治疗。但重要的步骤最好在早上进行，因为内源性皮质类固醇水平此时最高。长时间紧张的牙科治疗手术可能需要调

tal abscess, which did not respond to simple drainage and root surface debridement. The use of urinalysis strips (Fig 4-15b) within the surgery demonstrated high urinary glucose levels and a subsequent random blood glucose test (random rather than fasting glucose as patient had eaten within the previous eight hours) demonstrated marked hyperglycaemia.

In general, the better the diabetic control the better the response to periodontal therapy. Well-controlled diabetics can be treated as any other patient, but stressful procedures are probably best performed early morning, as endogenous corticoster-



a



b

图4-15　a. 1例未确诊糖尿病患者的多发性牙周外侧脓肿；b. 多项尿液分析条示例

Fig 4-15 (a) Multiple lateral periodontal abscesses in an undiagnosed diabetic.(b) Example of multi-test *urinalysis strips for in-surgery use.*

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节胰岛素或在治疗前调节升高血糖水平的片剂摄入方式。建议联络患者的主治医师。在糖尿病患者进行常规牙周治疗时没有使用抗生素的指征；但是在难治疗性病例中服用小剂量的多西环素可抑制PMNL胶原蛋白酶帮助伤口愈合。

oid levels are highest at this time. Prolonged stressful dentistry may require modification of insulin or tablet regimes to raise blood glucose levels prior to the therapy. Liaison with the patient's doctor is recommended. There is no indication for the use of antibiotics during routine periodontal therapy for diabetics; however, low dose doxycycline may inhibit PMNL collagenases and assist healing in refractory cases.

牙周炎血液病学危险因素

多种血液疾病能影响牙周组织。它们包括：

- 急性／慢性骨髓性白血病
- 骨髓发育不良综合征
- 粒细胞缺乏
- 周期性或良性／家族性中性粒细胞减少症
- 低／γ-球蛋白缺乏症（无丙种球蛋白血症）
- 淋巴细胞形成缺陷

这些疾病少见，最好参考潜在的出血性疾病和那些与不良PMNL功能相关的治疗。

Haematological risk factors for periodontitis

A variety of haematological diseases can affect the periodontal tissues. These include:

- acute/chronic myeloid leukaemia
- myelodysplasia syndrome
- agranulocytosis
- cyclical or benign/familial neutropenia
- hypo/agammaglobulinaemia
- defects of lymphocyte formation (Di George syndrome, Wiscott-Aldrich syndrome).

Such conditions are rare and best referred for advice/therapy due to potential bleeding problems and those related to poor PMNL function.

小 结

危险评估是现代牙周临床治疗的

Summary

Risk assessment is an important part of

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一个重要部分。发生牙周病的危险因素包括：

- 家族史
- 吸烟
- 内科疾病，如糖尿病等

牙周疾病进展的危险因素包括：

- 与年龄相关的现存附着丧失的严重程度
- 全口＞25％的位点探诊出血(BOP)
- 牙周袋深度＞4 mm
- 糖尿病控制不良
- 吸烟
- ＞30%的位点有菌斑
- 局部菌斑滞留因素
- 精神紧张
- （可能）营养不良

建议将系统性危险因素与疾病的诊断一起记录在病历上。

modern-day periodontal practice. Risk factors for acquiring periodontal disease include:

- family history
- smoking
- medical problems such as diabetes mellitus.

Risk factors for disease progression include:

- severity of existing attachment loss in relation to age
- whole mouth bleeding on probing at >25% of sites
- pockets >4mm in depth
- poorly controlled diabetes
- smoking
- plaque at >30% of sites
- local plaque-retention factors
- stress
- (possibly) poor nutrition.

It is recommended that systemic (patient-based) risk factors are documented alongside the diagnosis in patients case records.

拓展阅读
Further Reading

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第 5 章 牙周病的自然进程和临床体征

Natural History and Clinical Signs of Periodontal Diseases

目 的

本章的目的是阐明牙周炎自然进程的变化过程，帮助读者认识牙周健康状态和疾病状态重要的临床特征。

Aim

This chapter aims to explain the changing paradigm of the natural history of periodontitis and to help the reader visualise the important clinical features of periodontal health and disease.

要 点

读完本章后读者能清楚认识未治疗和治疗后的牙周疾病在进展方式上如何不同，能够认识患者牙龈疾病和牙周疾病的显著特征。

Outcome

The outcome of reading this chapter should be that the reader will understand how untreated periodontitis differs in its pattern of progression from treated disease, and will be able to recognise the salient features of gingival and periodontal diseases in their patients.

自然进程研究

疾病的自然进程是描述疾病在没有临床干预情况下随时间的进展发展变化的过程。自然进程的研究对于理解疾病如何运行，帮助制定健康护理计划和改善临床治疗结果是非常重要的。牙周病最终治疗结果是在舒适和

Natural History Studies

The natural history of a disease describes the pattern of behaviour of the disease process with time, in the absence of clinical intervention. Natural history studies are essential to our understanding of how a disease should behave and help to plan health

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功能状态下保留牙齿，这是成功的牙周护理最基本的结果衡量标准，但是，因为牙齿可由于许多原因而丧失，而且牙齿脱落的回顾性研究往往不能确定牙齿脱落的具体原因，因此很难进行衡量。所以常用到辅助的结果衡量标准。它们包括：

- 牙齿不松动或松动度与轻松舒适的功能相适应
- < 25% 的位点探诊出血
- 没有任何位点的牙周袋深度 > 4 mm
- 任意位点的龈沟内没有溢脓
- 菌斑计分范围在 20%～40% 或更低
- 稳定的或改善的 CALs

重要的是认识到必须在患者个体具体情况的基础上作出治疗结果是否理想的判断，因为 < 25% 的全口菌斑可能对某一个患者来说与其牙齿的稳定相适应，但对其他患者可能需要 < 10%。在衡量患者治疗成功的标准时除了以上所述外可能还要增加其他标准，比如：

- 最小的牙龈退缩
- 无过敏
- 美观

care and improve clinical outcomes. In periodontal diseases, the ultimate treatment outcome is the retention of teeth in a state of comfort and function for life. This is the primary outcome measure of successful periodontal care, but is very difficult to measure, as teeth may be lost for many reasons and retrospective studies of tooth loss are often unable to determine with certainty the cause of the tooth loss. Secondary outcome measures are therefore often used. These would include:

- lack of tooth mobility or mobility compatible with comfortable function
- BOP at < 25% of sites
- no pocketing > 4mm at any sites
- no suppuration from the gingival crevice at any sites
- plaque scores in the range of 20%–40% or less
- stable or improved CALs.

It is important to realise that decisions on ideal outcome goals must be made on an individual basis, since a target of <25% whole mouth plaque may be compatible with stability for one patient, but others may need to achieve levels <10%. Patients are likely to add to the above outcomes their own measures of success, such as:

- minimal recession
- no sensitivity
- good aesthetics

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• 无牙龈充血水肿
• 无臭味等

Harold Löe等，通过检查没有接受过口腔护理的斯里兰卡茶叶工人对其进行了一项经典的牙周病自然进程研究。这项研究对于帮助识别监控疾病危险因素尤为重要，该研究或者是横向的（在同一时刻在人群之间进行比较）或者是纵向的（纵向研究帮助联系原因和结果并能对疾病进展进行评估）。对斯里兰卡茶叶工人的研究持续了15年，发现他们的疾病发病率比挪威的对照组人群要高。尽管这些茶叶工人有大量的菌斑和牙石，但疾病的进展速度为：

8％的患者在牙周病快速进展组（高危组）

81％的患者以中等进展速度进展（中危组）

11％没有进展，明显有抗病能力

由于对疾病发生发展速度的定义方法的不一致，这些数据现在看来是较高，但是世界范围的研究仍然支持5％～15％成年人有遭受重度牙周疾病的危险。25岁以下具有严重牙周破坏危险的高加索人发生早发性（侵袭性）

• no gingival redness
• no malodour, etc.

Harold Löe and colleagues, who examined a population of Sri Lankan tea workers who had no access to dental care, performed one of the classical natural history studies. *Such population studies are important* in helping identify risk factors (Chapter 4) for the disease under scrutiny and are either cross-sectional (i.e. looking for comparisons between populations at one moment in time) or longitudinal (longitudinal studies help link cause to effect and allow disease progression to be assessed). The Sri Lankan tea-worker study took place over a 15-year period and found higher disease prevalence in the tea workers than a control population in Norway. Despite the fact that the tea workers had large amounts of plaque and calculus the progression rate for disease was:

8% in a rapid-progression group (high-risk group)

81% progressed at a moderate rate (medium-risk group)

11% showed no progression and were apparently disease resistant.

These figures are now regarded as being on the high side, owing to the methods used to define disease presence and progression, but, nevertheless, worldwide studies support figures of between 5% and 15% of the population being at risk from severe de-

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牙周疾病的统计数字接近1%，而对亚洲和加勒比黑人可高达3%。但是，Clerehugh等最近对167名年龄在14～19岁青少年进行了5年的研究，发现有77%的研究对象的附着丧失与高水平的菌斑和牙石有关。然而，当一小部分人群由于牙周炎而遭受全口牙列脱落的危险时，大部分人群可能遭受由于在较小年龄时发生的疾病而导致的部分牙齿脱落。牙周炎导致的失牙比例占35%。

struction in adult life. The statistics for early-onset (aggressive) diseases are closer to 1% of Caucasian subjects below the age of 25 years being at risk of significant destruction and up to 3% of Asian and Afro-Caribbean subjects. However, more recent studies by Clerehugh and colleagues have identified progression of attachment loss, albeit slow, in 77% of 167 adolescents aged 14–19 years at some point over a five-year observation period. The attachment loss was related to high levels of plaque and calculus. Nevertheless, whilst a minority of the population appears to be at risk of total tooth loss from periodontitis, a significant number may experience some tooth loss owing to disease that starts at a younger age than previously thought. Periodontitis is believed to account for 35% of all tooth loss.

线性疾病进展与突发理论

直到20世纪80年代早期，牙周疾病的进展被认为随时间呈直线方式进展（图5-1）。高危个体由于高水平的疾病活跃性比低危个体以较快速度丧失牙周附着。但是1982～1984年在波士顿的一系列研究改变了传统的范例，偶然暴发和多次不同时暴发假说随之产生（图5-2）。Goodson等调查了22名受试者1年，用人工探查每个牙6个位点，每2个月1次的方法。他们估计了

Linear Disease Progression Versus the Burst Theory

Until the early 1980s, periodontal disease progression was thought to follow a linear pattern with time (Fig 5-1). High-risk individuals would lose attachment at a rapid rate owing to a higher level of disease activity than lower-risk individuals. However, a series of studies performed between 1982 and 1984 in Boston changed the traditional paradigm and the *random burst and multiple asynchronous burst* hypotheses were

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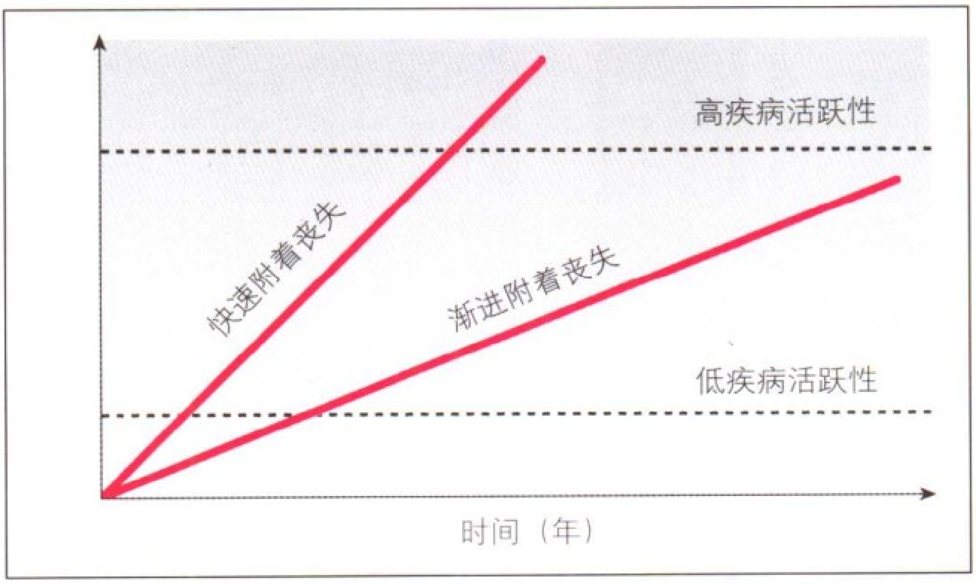


图 5-1 不同个体持续活跃的牙周病不同进展速度的“线性模型”

Fig 5-1 The “linear model” for periodontal disease progression assumes constant disease activity at different rates for different individuals

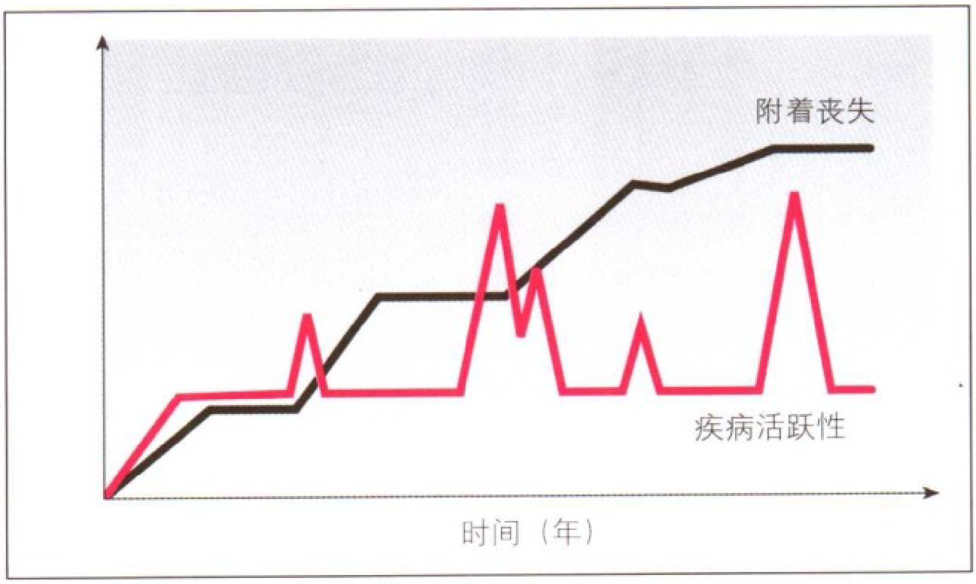


图 5-2 活跃期和静止期“随机或多次不同时暴发”模型

Fig 5-2 The “random burst or multiple asynchronous burst” model of Socransky and colleagues assumes periods of activity and remission. The latter vary in number and size, both between and within individuals

探诊时的误差，并对这个误差设定了阈值（大约 2.5 mm）来表示附着水平的真正变化。比较以前认为疾病的活跃期与非活跃期和修复期散在相间分

born (Fig 5-2). Goodson and colleagues examined 22 subjects by manual probing of six sites per tooth, every two months for a year. They estimated the errors involved

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布的思想，他们认为牙周病的进展是按照更随机偶然的方式进行，这些进展期数量和大小在个体间和个体受试者内均不同，有时以多次不同时暴发的方式出现。有人认为在这些研究中使用的高阈值有助于发现发生附着丧失的一个更不可预测的模式。1991 年亚拉巴马州的一个研究小组利用一台精确度达 0.1 mm（即非常小的变化）的电子探针检查研究 30 名受试者，发现 76% 的受试者为直线式疾病进展。但是他们使用了一个有争议的过低的阈值（29% 的位点发现活动性疾病——一个高统计数据），而且他们的分析方法支持直线模式。这些研究的结论是在有牙周炎倾向的人群中存在一个亚群，其疾病的进展是不连续的和不可预测的（约 25%）。

in their probing and set thresholds above this error (around 2.5mm) to signify a real change in attachment level. They determined that the disease process appeared to follow a more random pattern than previously thought where periods of active disease were interspersed with periods of inactivity and repair. The number and magnitude of episodes differed both between and within individual subjects, and sometimes occurred in multiple asynchronous bursts. Some claimed that the high thresholds used in these studies favoured the finding of a more unpredictable pattern of attachment loss. In 1991, a study by a group in Alabama utilised an electronic probe which measured to a degree of accuracy of 0.1mm (i.e. smaller increments) and found linear disease progression in 76% of their 30 subjects. However, they used a threshold arguably too low (active disease was found at 29% of sites – a high figure) and their analysis method favoured a linear model. The outcome of these studies was that there appeared to be a subgroup of the periodontitisprone population in whom disease progression was discontinuous and unpredictable (about 25%).

20世纪90年代的几项研究对这一知识体系进行了补充，他们使用了更大数量的受试个体和不同的研究设计来评估疾病进展模式。归纳起来，这些研究广义地分为两组：

Several studies during the 1990s added to the body of knowledge by assessing the pattern of disease progression using larger subject numbers and different study designs. Taken together, the studies broadly

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• 未治疗疾病的研究——证实暴发型进展模式

• 维持治疗期疾病的研究——显示直线型疾病进展模式

假如未经治疗的疾病存在许多促进性的局部危险因素和行为危险因素，上述结论似乎是合乎逻辑的，也能够期望一个无法预测的进展模式。一旦危险因素被纠正，致病因素（菌斑）被控制，就可能会期望任何疾病进展有可能按照更加可预测的模式进行。

疾病标志

这种随机暴发理论促进了许多致力于识别疾病活跃性标志的科研工作，它们优越于现存临床衡量标准。许多疾病标志可在龈沟液中检测到，对诊断（真正的阳性诊断）有 80% 的灵敏度和80%特异性（真的阴性诊断），对活跃性疾病发展的阳性或阴性的预测价值＞75%。同时它们比个别临床标准更加优越，但由于它们在牙科实践中过于麻烦，现在要把它们作为标准牙科业务是不可能的。本章将着眼于疾病的临床标志。重要的是记住，有经验的医师在决定一个患者或位点是否需要积极治疗前，他会考虑许多这类衡量标准。没有研究试图将诊断准

fall into two groups:

• Studies of un-treated disease – which demonstrate a burst type pattern of progress.

• Studies of treated disease during maintenance – which show a more linear pattern of disease progression.

The above outcome seems logical, given that untreated disease has many contributing local and behavioural risk factors, and one would thus expect an unpredictable pattern of progression. Once risk factors are corrected and causative agents (plaque) controlled, it maybe expected that any disease that progresses is likely to follow a more predictable pattern.

Disease Markers

The random burst theory stimulated a lot of research work aimed at identifying markers of disease activity, which were superior to existing clinical measures. Many such markers measured in GCF achieved 80% sensitivity for diagnosis (true positive diagnoses) and 80% specificity (true negative diagnoses), with positive and negative predictive values for the development of active disease >75%. Whilst these were far superior to individual clinical measures, they were too cumbersome for dental practice, and at present they look unlikely to become accepted as standard practice. This chapter will therefore focus on clini-

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确性的效果进行量化，因为有太多的变异性需要考虑，并且在这样的研究中需要的志愿者数目非常巨大。

cal markers of disease. It is important to remember that the experienced clinician takes a large number of such measures into account before deciding on whether a patient or site requires active therapy. No study has attempted to quantify the effectiveness of this approach in terms of diagnostic accuracy, because there are simply too many variables to account for, and volunteer numbers would need to be enormous in such studies.

原始牙周健康状态的临床体征

图 1-2 是一幅典型的正常牙周组织的图片。其特征有：

1. 粉红色牙龈（有个体差异；有的可表现由于种族差异引起的色素沉着）。
2. 致密坚韧的牙龈（无肿胀）。
3. 均匀一致的颜色（没有明显的红斑）。
4. 有点彩（"橘皮效应"）。
5. 刃状牙龈边缘。
6. 完整的龈缘（无溃疡）。
7. 平坦的锥体状牙间乳头（正常时充满邻间隙）。
8. 当用20～25g力量进行探诊时探诊深度＜3mm。
9. 无探诊出血。

Clinical Signs of Pristine Periodontal Health

Fig 1-2 is a picture of classical pristine periodontal health. Its features are:

1. Pink gingivae (there are individual differences; some may show racial pigmentation).
2. Firm gingivae (no swelling).
3. Uniform colour (no patches of marked redness).
4. Presence of stippling (referred to as "orange peel effect").
5. A knife-edged gingival margin.
6. Intact gingival margin (no ulceration).
7. A flat and triangular interdental papilla (normally filling inderdental space).
8. A probing depth < 3mm, when 20-25g weight is applied to the probe.
9. Complete absence of any bleeding to

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10. 无溢脓（脓液）。

11. 龈缘刚好位于 CEJ 上。

12. 无根分叉暴露。

13. 无牙龈退缩。

但重要的是要记住临床牙龈健康状态可能不典型（图1-9）。例如，牙分开有间隙时可有一变平的邻间龈乳头。另外由于牙刷的创伤和牙龈退缩而出现的附着丧失，虽然不是典型的健康状态，但可与健康状态一致。经牙周治疗后，可能出现牙周支持降低，暴露的根分叉，余留的漂移和松动度，但是疾病可不再是活跃的，所以组织处于临床健康状态（图5-3）。实际上，与年龄有关的变化如牙龈退缩和软组织连贯性的变化将导致老年人健康牙周组织很难达到典型的或"原始的"健康状态。

probe.

10. Complete absence of any suppuration (pus).

11. A gingival margin situated just above the CEJ.

12. Complete absence of furcation exposure.

13. No recession.

It is important to remember, however, that clinical gingival health may not be classical (Fig 1-9). For example, teeth spaced apart may have a flattened interdental papilla. In addition, attachment loss may occur owing to toothbrush trauma and recession may be consistent with health, albeit not classical health. Following periodontal therapy, there may be reduced support, exposed furcations, residual drifting and mobility, but the disease may no longer be active and hence the tissues are clinically healthy at that time (Fig 5-3). Indeed, age-related changes such as recession and changes in soft-tissue consistency result in healthy periodontal tissues in the



图5-3　治疗后保持7年临床健康患者，只需要偶尔去除牙石

Fig 5-3 Clinical health in a patient post-therapy who had been stable for seven years and required no more than occasional calculus removal.

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elderly failing to satisfy the conditions of classical or "pristine" health.

龈炎的临床体征

尽管龈炎并不总是发展为牙周炎，牙周袋的形成可在没有明显炎症的情况下产生（图5-4），但是大多数人都接受龈炎先于牙周炎发生的观点。炎症牙龈组织的典型特征是：

1. 充血（从龈乳头开始，沿龈缘进展）。
2. 钉突消失。
3. 肿胀（水肿）——肿胀时组织变得更软，按压时凹陷。
4. 龈缘卷缩邻间乳头失去三角形形状。
5. 轻探出血。

图5-5显示早期龈炎，而图5-6显示一个更慢性的确立的炎症状态，组织愈合导致纤维化。假性牙周袋随龈炎的加重而加深，牙周袋探诊深度超

Clinical Signs of Gingivitis

It is largely accepted that gingivitis precedes periodontitis, even though gingivitis does not always lead to periodontitis and periodontal pocketing can develop in the absence of obvious inflammation (Fig 5-4). However, the classical features of inflamed gingival tissues are:

1. Redness (starting at the papilla and progressing along the margin).
2. Loss of stippling.
3. Swelling (oedema) - with which the tissues become softer and "pit" on pressing.
4. Rolling of the gingival margin and loss of the triangular shape of the interdental papilla.
5. Bleeding on gentle probing.

Fig 5-5 shows early gingivitis, whereas Fig 5-6 demonstrates a more chronic and established inflammatory state, where the body's attempts at healing have led to



图5-4 深牙周袋形成伴轻微肉眼可见的炎症，非正规牙周探查很易忽略

Fig 5-4 Deep pocketing present, with very subtle signs of inflammation to the untrained eye. This would be easily missed unless routine periodontal probing is performed.

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过3 mm，但结合上皮仍在CEJ位置（图1-3）。

牙周炎的临床体征

已知牙周病的位点不一定表现所有龈炎体征，牙周炎的典型特征（图5-7）包括：

- 部分或所有龈炎体征（见前述）

fibrosis. False pocketing may arise with gingivitis, where the probing pocket depth exceeds 3mm, but the JE remains at the CEJ (Fig 1-3).

Clinical Signs of Periodontitis

Accepting that sites of periodontal disease may not necessarily demonstrate all the signs of gingivitis, classical features of periodontitis (Fig 5-7) are:

- some or all of the signs of gingivitis (see above)



图5-5　早期牙龈炎症，龈缘轻微红肿、点彩消失

Fig 5-5 Early signs of gingival inflammation, mild rolling of marginal tissues and loss of stippling.



图5-6　持续数月的慢性龈炎出现纤维化和水肿

Fig 5-6 Chronic gingivitis of several months' duration, with fibrosis and oedema present.

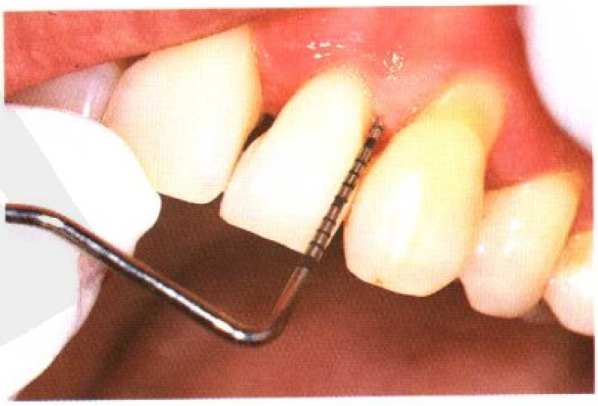


图 5-7　UNC-5 探针测定典型牙周炎

Fig 5-7 Classical periodontitis measured using University of North Carolina 15mm (UNC-15) probe.

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- 真性牙周袋，即牙周袋探诊深度≥4 mm，牙周袋底位于CEJ根方（即附着丧失产生）
- 牙龈退缩
- 牙周溢脓
- 牙齿动度大于生理水平（>0.2mm）
- 牙齿漂移
- 根分叉暴露
- 骨吸收的X线检查证据

- true pocketing on probing, i.e. probing pocket depths ≥ 4mm, where the pocket base is apical to the CEJ (i.e. attachment loss has occurred)
- recession
- suppuration
- mobility above physiological levels (> 0.2mm)
- drifting of teeth
- exposure of furcations
- radiographic evidence of bone loss.

活动性疾病经历

牙周病的临床标准主要的局限是他们衡量疾病病程而不是衡量疾病当前活跃性。例如，放射检查显示的骨丧失和临床附着丧失为牙周破坏的累积效果提供了到当前时间为止的衡量标准。确定一个位点是活动性或非活动性的重要性是指对非活动性部位的器械操作会因器械操作创伤导致附着丧失。所以判断是否需要一个位点一个位点地进行治疗是非常重要的，同时需要考虑该位点所有有价值的信息。

Historical Verses Active Disease

The principal limitation of clinical measures of periodontal disease is that they measure disease experience rather than current disease activity. For example, radiographic bone loss and clinical attachment loss provide measures of the cumulative effects of periodontal damage, up to a point within the recent past. The importance of determining whether a site is active or inactive is that instrumentation of inactive sites can lead to attachment loss due to the trauma of instrumentation. It is important therefore to determine the need for therapy on a site-by-site basis, taking into account all available information for that site.

示例 1

6 mm深的牙周袋无探诊出血和溢脓且不能探查到龈下沉积物时，不应该进行器械操作，在4～6个月后复诊。

Scenario 1

A 6mm pocket that does not bleed when probed nor exhibit pus discharge,and within which subgingival deposits cannot be

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如果6 mm的牙周袋保持稳定，可能只需进行龈下预防保健。

detected, should not be instrumented, but simply reviewed after four to six months. If the 6mm pocket remains stable, subgingival prophylaxis may be the only therapy indicated.

示例 2

6 mm 的牙周袋表现如示例 1，但能探测到龈下牙石，应该进行龈下刮治并在 3～4 个月后复诊。

Scenario 2

A 6mm pocket presenting as for scenario 1, but with detectable subgingival calculus, should be scaled subgingivally and reviewed after three to four months.

示例 3

6 mm 的牙周袋表现如示例 1，但伴有探诊出血，应进行牙根清创，并在 3～4 个月后复诊。

Scenario 3

A 6mm pocket presenting as in scenario 1, but with bleeding on probing, should be root debrided and reviewed three to four months later.

在所有的病例，都应谨慎地保证没有局部龈下危险因素存在。

In all cases, care must be taken to ensure that no subgingival local risk factors exist.

位点特异性（特殊性）的概念

要考虑的与健康／疾病状态的临床体征有关的最后一个概念是位点特异性，简单的意思就是指微生物群、解剖、局部免疫反应等。牙齿周围的一个位点可能与同一牙上相邻的一个位点完全不同。这最好用一个免疫印迹检测板来说明（图5-8）。免疫印迹检测板是一种科研工具，它利用 DNA 探针与菌斑样品互补的细菌 DNA 进行交叉结合来测定需要鉴定的许多细菌种类。在图5-8中，每一垂直列代表一种特定的微生物，每一水平排代表一个独立

Concept of Site-Specificity

The final concept to consider in relation to clinical signs of health/disease and patterns of disease progression is that of site-specificity. This simply means that the micro-flora, the anatomy, the local immune response, etc. at one site around one tooth, may be totally different from an adjacent site of the same tooth. This is best illustrated with a checkerboard immunoblot (Fig 5-8). The immunoblot is a research tool which uses DNA probes to as many species as necessary and cross-binds the probes

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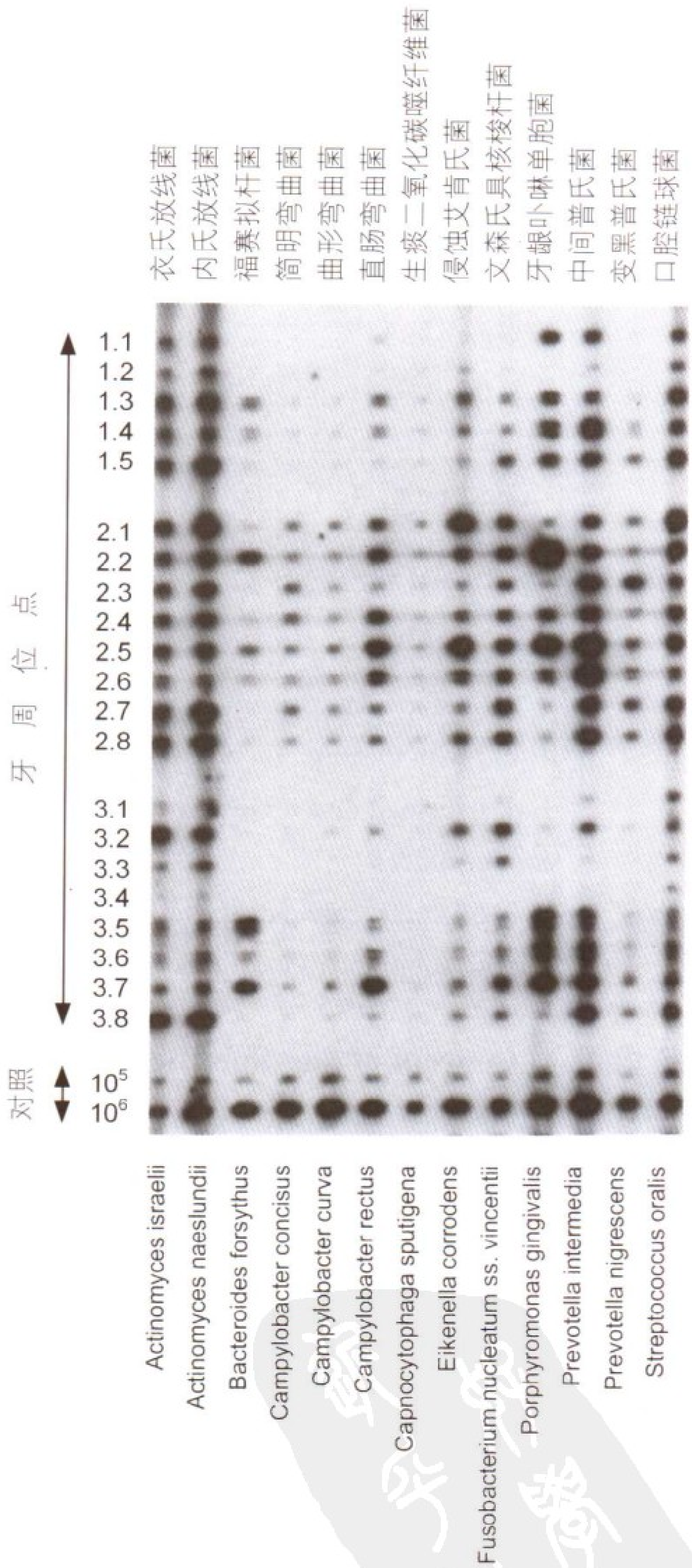


图 5-8　免疫印迹法测定同一患者不同位点的微生物

Fig 5-8　Checkerboard immunoblot showing which organisms were detected at individual sites within the same patient.

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位点的一个菌斑样本。一个黑色斑点出现的地方，表示该位点有特定的微生物存在。图中牙1.1的位点的印迹外观与同一患者同一牙的1.3位点有非常大的区别。这一关联关系说明牙周治疗必须依据从详细记录图表获取的位点特异性数据而不是依据"全口"外观（见第8章）。

to the complementary bacterial DNA in a plaque sample. In Fig 5-8, each vertical column represents a particular organism and each horizontal row represents a plaque sample from a single site. Where a black spot appears, the organism is present at that site. The profile for the site sampled on tooth 1.1 is very different from that for 1.3, from the same tooth in the same patient. The relevance of this is that periodontal therapy must be based upon site-specific data derived from detailed charts and not on "whole mouth" appearances (see Chapter 8).

拓展阅读
Further Reading

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2 Goodson JM, Tanner AC, Haffajee AD, Sornberger GC, Socransky SS. Patterns of progression and regression of advanced destructive periodontal disease. J Clin Periodontol, 1982;9:472–481

3 Löe H, Anerud A, Boyson H, Smith M. The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. J Periodontol, 1978; 49:607–620

4 Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. J Clin Periodontol, 1984;11:21–32

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第6章 牙周病分类

Classification of Periodontal Diseases

目　的

本章目的是阐述分类系统的必要性和该系统是如何作为牙周病学的一部分发展至今。将重点阐述分类系统的缺点，并给普通牙科治疗提出一种简化易懂的分类。

Aim

This chapter aims to explain the necessity for classification systems and how such systems have evolved in periodontology to the present day. The faults with existing systems will be highlighted and a simplified classification proposed for general dental practice.

要　点

通过本章的学习，临床医师能够明白牙周病学中旧的和现行的诊断术语间的年代关系，并能将一种分类逻辑系统应用于临床实践中。

Outcome

The outcome of reading this chapter should be that the practitioner understands the chronological relationship between old and current diagnostic terms in periodontology, and is able to introduce a logical system of classification into their own clinical practice.

为什么我们需要分类系统?

在概念方面，本章是本书中最简单的部分，但却证明是最难写的，因为关于牙周病分类系统存在着广泛的争论。

Why Do We Need Classification Systems?

This chapter is the simplest in the book in terms of concepts, but proved the hardest to write, owing to the broad controversy that surrounds classification systems for

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periodontal diseases.

传统的分类系统比较简单。过去由于在科研中缺乏认识和证据，对这些疾病的认识较少，所以，只从必要性方面出发使用了简单的术语。在20世纪70年代，80年代和90年代早期，对牙周病的研究激增，我们的认识基础显著扩大起来。所以在20世纪70年代，80年代和90年代早期的分类类型被扩增，并包括了新的疾病——主要是基于病例报道有公认证据的疾病。但是在认识和理解之间存在重大区别。结果（就像现代社会大多数事物一样），转了一圈，又回到原位，1999 年国际牙周病分类专题讨论会又重新减少了普通疾病类型的数目，这反映了我们缺乏对这些复杂疾病真正的理解以及用什么来鉴别它们，并增加了不寻常疾病类型的数目。

Traditionally, classification systems were simple. Owing to a paucity of knowledge and evidence from research, little was known about these diseases and thus simple terms were applied, by necessity. During the 1970s, 1980s and early 1990s, research into periodontal diseases exploded and our knowledge base broadened dramatically. The classification categories of the 1970s, 1980s and early 1990s therefore expanded to encompass new diseases for which there was putative evidence, based largely on case reports. However, there are important differences between having knowledge and having understanding. As a consequence (as with most things in the modern world), the wheel has turned almost full circle and the 1999 International Workshop on Periodontal Disease Classification has re-reduced the number of common disease categories – reflecting our lack of genuine understanding of these complex diseases and what differentiates them – and increased the number of less-common disease categories.

分类系统是必不可少的，主要是因为如果没有它就不可能得出诊断。没有一个明确的诊断，就不可能完成恰当的治疗。分类系统的作用在于：

- 指导人们认识更多该疾病涉及内容的科学研究。

Classification systems are essential, largely because without them it is not possible to arrive at an informed diagnosis. Without a robust diagnosis, appropriate therapy cannot be implemented. Classification systems also:

- Direct research aimed at learning more about the diseases concerned.

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- 帮助确认更好治疗目标的证据基础。
- 引导临床医师获得治疗疾病的最佳方法。
- 使国际社会能够用共同的语言进行交流。
- 引导公共健康计划和治疗目标。
- 帮助临床医师制定治疗方案使患者最大受益。

- Help determine the evidence base for better-targeted therapy.
- Guide practitioners towards the best method for treating a disease.
- Enable the international community to communicate in a common language.
- Guide public health planning and targeting of therapy.
- Help practitioners plan treatment protocols to maximise benefit to all their patients.

本系统内的基本术语

当设计分类系统时，一系列传统术语常被用作亚分类的基础。

Basic Terms that Appear in Such Systems

When designing classification systems, a series of traditional terms is often used as the basis for sub-categorisation.

急 性

"急性"是在疾病分类中最常用的一个术语。术语"急性"描述一种短期的、剧烈的、活跃的过程，常常是一个使人痛苦的需要采取即刻行动的术语。现在的分类已经从疾病如AUG名称中去除掉，AUG变成ANUG，现在又被NUG取代。同样的情形出现在"急性外侧牙周脓肿"，现在称为"外侧牙周脓肿"。急性疾病常常是可治愈的，例如儿童急性成淋巴细胞性/细胞性白血病。去除感染病因能够治愈大部分急性感染。

Acute

The term "acute" is one of the most frequently used in medical disease classification. "Acute" is a term that describes a short-lived, intense, brisk experience that is often a distressing one and is assumed to require immediate action. Recent classifications have thus removed it from the titles of diseases like AUG, which became ANUG, but has now been replaced by the term NUG. The situation is similar for the acute lateral periodontal abscess, now called a "lateral periodontal abscess". Acute diseases may often be curable, e.g. acute lymphoblastic/cytic leukaemia in children.

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Eliminating the infective agent can cure the majority of acute infections.

慢 性

如果疾病不是急性的，则认为它是一个慢性过程。"慢性"疾病有一个较长的"时间过程"，并且通常不会立即构成生命威胁。然而，指明一个疾病急性或慢性的"时间过程"的证据基础在不同系统之间是不同的，在牙周膜内对于什么可能是长或短的时间过程没有一致意见。慢性病通常不能治愈，其治疗包括症状控制或更严重的并发症的预防。例如，糖尿病和艾滋病的治疗是预防系统性并发症和尽可能地延长寿命使之接近正常。风湿性关节炎和克隆病的治疗是根据症状进行治疗。牙周疾病大部分是慢性的，现在无法治愈，但能被控制。

Chronic

If a disease is not acute, then by default it is a chronic process. "Chronic" diseases have a longer "time course" and are generally not immediately life-threatening. However, the evidence base for the "time course" that designates a disease acute or chronic varies from one human system to the next, and in the periodontium there is no consensus on what a short or long time course may be. Chronic diseases are generally incurable, and management involves the control of symptoms, or the prevention of more serious complications. For example, diabetes mellitus and HIV disease are managed to prevent systemic complications and prolong life-expectancy to as close to normal as possible. Rheumatoid arthritis and Crohn's disease are managed symptomatically. Periodontal diseases are largely chronic and cannot currently be cured, but they can be controlled.

侵袭性

"侵袭性"是描述在一个短时间内（数月或数年）的一个快速进展过程的术语，以前所用到的"快速进展性"就是这种方式。

Aggressive

"Aggressive" is a term that describes a rapid progression over a short period of time (months or a few years). Previously the term "rapidly progressing" was used in this manner.

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青少年性

"青少年性"是用来描述在青少年时期发生的疾病。

Juvenile

"Juvenile" is a term used to describe a disease whose onset occurred during juvenile years.

局限性

"局限性"是用来指明涉及一小部分牙齿的疾病（见下述）。

Localised

"Localised" is a term used to designate a disease involving a small number of teeth (see below).

广泛性

"广泛性"是用来描述涉及口内存在的大部分牙齿的疾病。

Generalised

"Generalised" is the term used to describe diseases involving the majority of teeth present in the mouth.

1999 年前的历史观点

多年来许多不同术语被用来描述牙周疾病。在 Wilfred Fish 爵士 1952 年的教科书《牙周病：治疗手册和病理学图谱》第 2 版中，该疾病被分类为：

退化／生长异常——包括老年性牙槽骨吸收（骨质疏松变化），牙骨质破坏（根尖吸收）和牙骨质增生。

炎症——包括创伤性炎症，急性溃疡性口炎（现在称 NUG），亚急性边缘性龈炎，慢性边缘性龈炎（包括药物性增生），单纯的溢脓（轻度牙周炎）和深度溢脓

Historical Perspective pre-1999

Many different terms have been used for periodontal diseases over the years. In Sir Wilfred Fish's 1952 text, *Parodontal Disease: A Manual of Treatment and Atlas of Pathology* (2nd edn.), the diseases were classified as:

Degenerative/abnormalities of growth - these included senile alveolar resorption (osteoporotic changes), odontoclasia (root apex resorption) and hypercementosis.

Inflammations - these included traumatic inflammations, acute ulcerative stomatitis (now called NUG), subacute marginal gingivitis, chronic marginal gingivitis (which included drug-

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（重度牙周炎）。

瘤形成——包括破牙质细胞瘤、牙骨质瘤和纤维龈瘤。

从现在国际分类系统（表6-1和6-2）可看出有一些在50年间改变很少！

表 6-1 阐述了多年来分类系统的变化。图6-1中阐明了侵袭性牙周炎发展的内在关系。此术语学导致了许多混淆，所以当美国牙周病学研究院和欧洲专题研讨会在20世纪90年代再次提出不同的系统时，得到世界范围内的认同。"牙周病分类国际专题研讨会"得以成立并在1999年11月作出报告，他们现在的观点是基于最可靠的数据基础上的。表6-2和6-3阐明了这一观点，但却非常复杂，部分是按照美国和部分欧洲健康保险公司的要求，为确定治疗费用而用专用名词。

induced overgrowths), pyorrhoea simplex (mild periodontitis) and pyorrhoea profunda (advanced periodontitis).

Neoplasia–which included odontoclastoma, cementoma and fibrous epulis.

It can be seen from the current international system (Tables 6-1 and 6-2), how little some things have changed in fifty years!

Table 6-1 illustrates the changes in classification systems over the years. The evolutionary inter-relationships of the aggressive periodontal diseases are illustrated in Fig 6-1. Such terminology has led to a great deal of confusion and when the American Academy of Periodontology and a European Workshop produced differing systems again in the 1990s, it was time for worldwide agreement. The "International Workshop for Classification of Periodontal Diseases and Conditions" was therefore established and reported in December 1999, their current view based on the best evidence available to date. This is illustrated in Tables 6-2 and 6-3, but is unhelpfully complex, being driven, in part, by the needs of health insurance companies in the USA and parts of Europe to have designated terms against which fees can be arranged.

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表 6-1 历年牙周病分类系统
Table 6-1 Classification systems through the years.

时间	责任机构	分类系统
1977	美国牙周病学研究院	青少年牙周炎 慢性边缘性牙周炎
1986	美国牙周病学研究院	青少年牙周炎（青春前期） （局限性）（广泛性） 成年性牙周炎 坏死溃疡性牙周炎 难治性牙周炎
1989	J Lindhe 编著，临床牙周病学，第2版，Munksgard：哥本哈根，1989	轻度牙周炎 重度牙周炎
1989	美国牙周病学研究院	早发性牙周炎 成年性牙周炎 与系统性疾病有关的牙周炎 坏死溃疡性牙周炎 难治性牙周炎
1993	欧洲牙周病学研讨会	早发性牙周炎 成年性牙周炎 坏死溃疡性牙周炎
1999	世界专题研讨会	见表 6-2 和表 6-3

表 6-2 1999 国际专题研讨会——牙龈疾病
Table 6-2 International workshop 1999 - gingival diseases.

A．菌斑性牙龈疾病	**A. Dental plaque-induced gingival diseases**
1．只与菌斑有关的牙龈炎	*1. Gingivitis associated with dental plaque only*
a．没有其他局部促进因素	a. without other local contributing factors
b．伴局部促进因素（I.1 表 6-3）	b. with local contributing factors (I.1 Table 6-3)
2．受系统因素影响的牙龈疾病	*2. Gingival diseases modified by systemic*

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（续　表）

	factors
a. 与内分泌系统相关的	a. associated with the endocrine system
1）青春期龈炎	1) puberty-associated gingivitis
2）月经周期性龈炎	2) menstrual cycle-associated gingivitis
3）妊娠期	3) pregnancy-associated
a）龈炎	a) gingivitis
b）化脓性肉芽肿	b) pyogenic granuloma
4）糖尿病相关性龈炎	4) diabetes mellitus-associated gingivitis
b. 与血液疾病相关的	b. associated with blood dyscrasias
1）白血病性龈炎	1) leukemia-associated gingivitis
2）其他	2) other
3. 药物相关性牙龈疾病	*3. Gingival diseases modified by medications*
a. 药物性牙龈疾病	a. drug-influenced gingival diseases
1）药物性牙龈肥大	1) drug-influenced gingival enlargements
2）药物性龈炎	2) drug-influenced gingivitis
a）口服避孕药性龈炎	a) oral contraceptive-associated gingivitis
b）其他	b) other
4. 营养不良性龈疾病	*4. Gingival diseases modified by malnutrition*
a. 抗坏血酸缺乏性龈炎	a. ascorbic acid-deficiency gingivitis
b. 其他	b. other
B. 非菌斑性牙龈疾病	**B. Non-plaque-induced gingival lesions**
1. 特异性细菌引起的牙龈疾病	*1. Gingival diseases of specific bacterial origin*
a. 淋球菌相关性损害	a. Neisseria gonorrhea-associated lesions
b. 梅毒密螺旋体相关性损害	b. Treponema pallidum-associated lesions
c. 链球菌相关性损害	c. streptococcal species-associated lesions

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（续　表）

d．其他	d. other
2．病毒引起的牙龈疾病	*2. Gingival diseases of viral origin*
a．疱疹病毒感染	a. herpes virus infections
1）原发性疱疹性龈口炎	1) primary herpetic gingivostomatitis
2）复发性口腔疱疹	2) recurrent oral herpes
3）水痘带状疱疹病毒感染	3) varicella-zoster infections
b．其他	b. other
3．真菌引起的牙龈疾病	*3. Gingival diseases of fungal origin*
a．念珠菌感染	a. Candida-species infections
1）广泛性牙龈念珠菌病	1) generalized gingival candidosis
2）线形牙龈红斑	2) linear gingival erythema
3）组织胞浆菌病	3) histoplasmosis
4）其他	4) other
4．遗传性牙龈疾病	*4. Gingival lesions of genetic origin*
a．遗传性牙龈纤维瘤病	a. hereditary gingival fibromatosis
b．其他	b. other
5．系统疾病的牙龈表现	*5. Gingival manifestations of systemic conditions*
a．皮肤黏膜疾病	a. mucocutaneous disorders
1）扁平苔藓	1) lichen planus
2）类天疱疮	2) pemphigoid
3）寻常型天疱疮	3) pemphigus vulgaris
4）多形红斑	4) erythema multiforme
5）红斑狼疮	5) lupus erythematosus
6）药物性	6) drug-induced
7）其他	7) other
b．变态反应	b. allergic reactions
1）牙科修复材料	1) dental restorative materials
i）汞	i) mercury
ii）镍	ii) nickel
iii）丙烯酸类树脂	iii) acrylic

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（续　表）

iv）其他	iv) other
2）引起反应的	2) reactions attributable to
i）牙膏／牙粉	i) toothpastes/dentifrices
ii）漱口液／冲洗液	ii) mouthrinses/mouthwashes
iii）口香糖添加剂	iii) chewing gum additives
iv）食物和添加剂	iv) foods and additives
3）其他	3) other
6．创伤性病损（人为的、医源性的、意外的）	*6. Traumatic lesions (factitious, iatrogenic, accidental)*
a．化学损伤	a. chemical injury
b．物理损伤	b. physical injury
c．温度损伤	c. thermal injury
7．异物反应	*7. Foreign body reactions*
8．非另外特别说明的（NOS）	*8. Not otherwise specified (NOS)*

表 6-3　1999 年国际专题研讨会——牙周疾病
Table 6-3 International workshop 1999 – periodontal diseases.

C．慢性牙周炎	**C. Chronic periodontitis**
1．局限性	*1. Localised*
2．广泛性	*2. Generalised*
D．侵袭性牙周炎	**D. Aggressive periodontitis**
1．局限性	*1. Localised*
2．广泛性	*2. Generalised*
E．系统性疾病的临床表现——牙周炎	**E. Periodontitis as a manifestation of systemic diseases**
1．与血液疾病相关的牙周炎	*1. Associated with haematological disorders*
a．获得性中性粒细胞减少症	a. Acquired neutropenia
b．白血病	b. Leukemias

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（续　表）

c．其他	c. Other
2．与遗传性疾病相关的牙周炎	*2. Associated with genetic disorders*
a．家族性周期性粒细胞减少症	a. Familial and cyclic neutropenia
b．唐氏综合征	b. Down syndrome
c．白细胞黏附缺陷综合征	c. Leucocyte adhesion deficiency syndromes
d．掌跖角化—牙周破坏综合征	d. Papillon-Lefevre syndrome
e．Chediak-Higashi 综合征	e. Chediak-Higashi syndrome
f．组织细胞增多综合征	f. Histiocytosis syndromes
g．糖原贮藏疾病	g. Glycogen storage disease
h．婴儿遗传性粒细胞缺乏症	h. Infantile genetic agranulocytosis
i．Cohen 综合征	i. Cohen syndrome
j．Ehlers-Danlos 综合征（Ⅳ型和Ⅷ型）	j. Ehlers-Danlos syndrome (Types IV and VIII)
k．低磷酸酯酶血症	k. Hypophosphatasia
l．其他	l. Other
3．非另外特别说明的（NOS）	*3. Not otherwise specified (NOS)*
F．坏死性牙周疾病	**D. Necrotising periodontal diseases**
1．坏死溃疡性龈炎（NUG）	*1. Necrotising ulcerative gingivitis (NUG)*
2．坏死溃疡性牙周炎（NUP）	*2. Necrotising ulcerative periodontitis (NUP)*
G．牙周脓肿	**E. Abscesses of the periodontium**
1．牙龈脓肿	*1. Gingival abscess*
2．牙周脓肿	*2. Periodontal abscess*
3．冠周脓肿	*3. Pericoronal abscess*
H．与牙髓疾病相关的牙周炎	**F. Periodontitis associated with endodontic lesions**
牙周－牙髓联合病变	*Combined periodontic-endodontic lesions*
I．发育性或获得性异常	**G. Developmental or acquired deformities and conditions**

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（续　表）

1．促进菌斑性牙龈疾病／牙周炎的局部牙相关因素	*1. Localised tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis*
a．牙体解剖因素	a. Tooth anatomic factors
b．牙科修复体／矫正器	b. Dental restorations/appliances
c．根折	c. Root fractures
d．牙根颈部吸收和牙骨质裂	d. Cervical root resorption and cemental tears
2．牙齿周围膜龈异常	*2. Mucogingival deformities and conditions around teeth*
a．牙龈／软组织退缩	a. Gingival/soft tissue recession
1）唇或舌面	1) facial or lingual surfaces
2）邻面（龈乳头）	2) interproximal (papillary)
b．角化龈不足	b. Lack of keratinized gingiva
c．前庭沟变浅	c. Decreased vestibular depth
d．异常的系带／肌肉位置	d. Aberrant frenum/muscle position
e．牙龈过量	e. Gingival excess
1）假性牙周袋	1) pseudopocket
2）不协调的龈缘	2) inconsistent gingival margin
3）牙龈增生	3) excessive gingival display
4）牙龈肥大	4) gingival enlargement
f．颜色异常	f. Abnormal colour
3．无牙牙槽嵴上膜龈异常	*3. Mucogingival deformities and conditions on edentulous ridges*
a．垂直和（或）水平向牙槽嵴不足	a. Vertical and/or horizontal ridge deficiency
b．牙龈／角化组织缺乏	b. Lack of gingiva/keratinised tissue
c．牙龈／软组织肥大	c. Gingival/soft tissue enlargement
d．异常系带／肌肉位置	d. Aberrant frenum/muscle position
e．前庭沟变浅	e. Decreased vestibular depth
f．颜色异常	f. Abnormal colour

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（续　表）

4．殆创伤	*4. Occlusal trauma*
a．原发性殆创伤	a. Primary occlusal trauma
b．继发性殆创伤	b. Secondary occlusal trauma

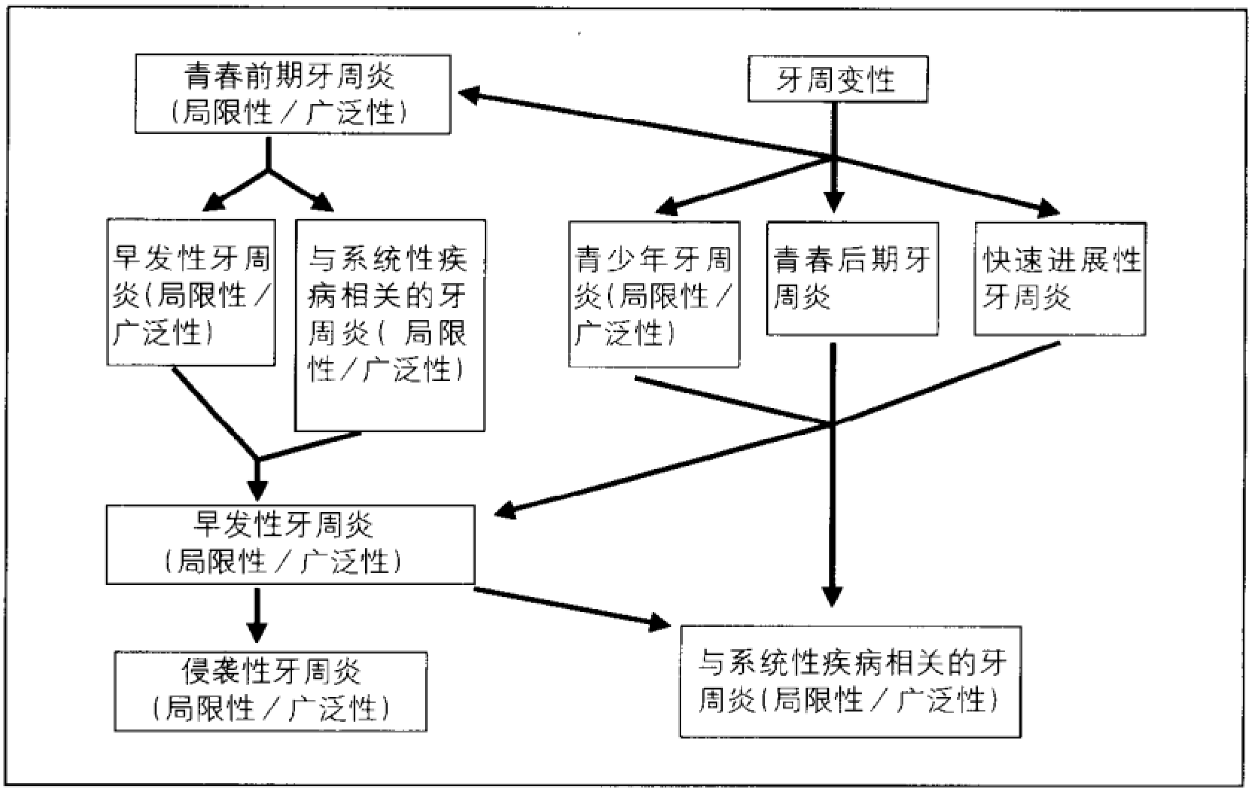


图 6-1　近年用于描述侵袭性牙周炎的不同术语间的内在联系

Fig 6-1 The inter-relationship between various terms used to describe aggressive forms of periodontitis over the years.

牙周疾病和状况的国际专题研讨会

International Workshop for Classification of Periodontal Diseases and Conditions

1999年的牙周病分类尽管在常规口腔治疗中过于麻烦，且仍然存在争论，但已逐渐被接受作为一种有用的参照系统。它的目的是指出1989年的

The 1999 classification is still controversial, but is becoming accepted as a useful reference system, albeit too cumbersome for day-to-day general dental practice.

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分类系统的局限性，包括：

- 没有对牙龈疾病进行分类。
- "成年性牙周炎"这个术语暗示慢性牙周附着丧失只发生在成年人，但是有证据表明在青少年同样会发生慢性牙周附着丧失（第 5 章）。
- "早发性牙周炎"是指适用于<35岁的牙周病个体的一个术语，而侵袭性疾病也能影响年龄稍大的个体。
- 与系统性疾病相关的牙周炎—用许多实际上是系统疾病在牙周组织上的表现来做不适当的定义。

1999年的分类系统通过下面对以前的分类系统的调整来强调了上述问题：

- 提出了牙龈疾病的类型。
- 用"慢性"代替了"成年性"。
- 难治性牙周炎这一独立类型被取消，因为所有形式的牙周炎都可能反复发作，但这并不一定意味存在着有共同生物学危险因素而使它们对抗治疗的个体群。

It aimed to address some of the limitations of the 1989 system, which included:

- No category for gingival diseases/conditions.
- The term "adult periodontitis", implying that chronic periodontal attachment loss only affected adults, although there is good evidence that chronic attachment loss can affect adolescents (Chapter 5).
- Early-onset periodontitis—a term applied to disease affecting individuals <35yrs of age, even though aggressive disease can affect slightly older individuals also.
- Periodontitis associated with systemic disease—poorly defined with many of the conditions in fact periodontal manifestations of systemic diseases.

The 1999 system therefore addressed the issues above by making the following changes to previous systems:

- A category for gingival diseases was introduced.
- The term "adult" was replaced by "chronic".
- Refractory periodontitis was removed as a separate entity, since it was felt that all forms of periodontitis may rebound as recurrent disease, but that did not necessarily mean that a group of individuals existed with common bio-

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logical risk factors that made them resistant to therapy.

- 用"侵袭性"代替了"早发性"。
- 能影响牙周组织的系统疾病被更全面的定义。
- "坏死性牙周疾病"被用来覆盖NUG和NUP，因为并不清楚这两种疾病是否是独立的疾病。
- 增加了牙周脓肿和牙周牙髓联合病变。
- 增加了"发育性"或"获得性"疾病状况。

- The term "aggressive" was introduced to replace the term "early-onset".
- Systemic diseases that can affect the periodontal tissues were more comprehensively defined.
- The term "necrotising periodontal diseases" was adopted to cover NUG and NUP, since it was unclear whether these two were separate disease entities.
- Periodontal abscess and periodontal-endodontic lesions were added.
- A category for "developmental" or "acquired" conditions was also added.

1999年分类系统的特殊问题

用"慢性"代替"成年性"

对先前陈述的理由来说这似乎是符合逻辑的。"慢性"指的是未治疗疾病随时间而进展的速度，而不是说疾病是不能治疗的。治疗的目的是稳定和再生，而不是治愈疾病。重要的是患者认识到牙周病不能治愈，只能控制，认识到他们在日常治疗过程中所起的关键作用。每个个体的进展速度依赖于局部和全身危险因素。许多患者在接受指导后，就能控制如吸烟等外来危险因素。普通术语中：

Specific Issues with the 1999 System

Replacement of "adult" with 'chronic"

This move seems logical for reasons previously stated. The term "chronic" refers to the rate of progression of *untreated* disease with time and does not imply that the disease is untreatable. Treatment is aimed at stabilisation and regeneration, rather than curing the disease. It is important that patients realise that periodontal diseases cannot be cured, only controlled, and their role in that process is crucial on a day-to-day basis. The rate of progression in each individual depends upon local and systemic risk factors. External risk factors like smoking

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局限性疾病影响< 30% 的位点
广泛性疾病影响> 30% 的位点

轻度疾病涉及 1～2 mm CAL
中度疾病涉及 3～4 mm CAL

重度疾病涉及> 5 mm CAL

复发性疾病与难治性疾病

难治性疾病习惯性地被认为是复发性疾病，发生在得到好的家庭护理和适当治疗的个体。在1999年的分类系统中"复发性疾病"适用于任何疾病类型，且发生的可能性受到如下因素的影响：

- 疾病程度
- 根分叉暴露
- 牙齿类型和位置
- 殆创伤
- 菌群
- 宿主反应
- 吸烟等

"复发性"的正确含义是指曾患有牙周疾病的任何人如果没有保持菌斑控制均可再次发生该疾病。

侵袭性牙周炎

指的是不论年龄大小，全身健康的牙周炎患者在短期内出现CAL。实

can be controlled by many patients when educated. In general terms:

Localised disease affects <30% of sites
Generalised disease affects >30% of sites
Mild disease involves 1–2mm CAL
Moderate disease involves 3–4 mm CAL
Severe disease involves >5mm CAL.

Recurrent disease v. refractory

Refractory disease was traditionally regarded as recurrent disease in individuals who had good home care and had been treated adequately. The term "recurrent disease" in the 1999 system can be applied to any disease category and the likelihood of it occurring is affected by factors such as:

- extent of disease
- furcation exposure
- tooth type and position
- occlusal trauma
- microflora
- host response
- smoking, etc.

The term "recurrent" implies, correctly, that anyone who has had periodontal disease can redevelop the disease if plaque control is not maintained.

Aggressive periodontitis

This term implies CAL over a short period of time irrespective of age, but in pa-

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际上，疾病初次出现和有潜在的生物学易感性的大多数个体都是年轻人，其特征包括：

- 全身健康。
- 家族集聚性。
- 快速的附着丧失和骨丧失。
- 组织破坏程度与菌斑水平不一致。
- *Aa*比例上升，在部分病例*Pg*可能升高。
- 可能存在异常的受到抑制的吞噬细胞功能。
- 可能存在异常升高的吞噬细胞功能。
- 这种疾病状况可"突然停止"。

局限性侵袭性牙周炎（图6-2）的特征有：

- 青春期前后发病。
- 细菌所致的强血清抗体反应（但反应可能是无效的）。
- 局限在第一磨牙和切牙，伴有2个或2个以上牙齿的邻面附着丧失，且不累及2个以上非第一磨牙或切牙。

广泛性侵袭性牙周炎（图6-3）的特征有：

- 患者年龄常＜30岁（也可见年龄更大者）
- 对感染微生物的血清抗体反应

tients who are systemically healthy. In practical terms, the majority of such individuals are young when the disease first manifests and underlying biological susceptibility is present. Key characteristics include:

- patients are systemically healthy
- familial aggregation
- rapid rate of attachment and bone loss
- tissue destruction is inconsistent with levels of plaque
- *Aa* is elevated in proportion and *Pg* may be in some cases
- there may be abnormally suppressed phagocyte function
- there may be abnormally raised phagocyte function
- the condition may "burn out".

Features specific to *localised* aggressive periodontitis (Fig 6-2) are:

- circumpubertal onset
- robust serum antibody response to bacteria involved (but response may be ineffective)
- localised to 1st molar and incisor teeth, with interproximal attachment loss at 2 or more teeth and not involving more than 2 non-lst molar or incisor teeth.

Features specific to *generalised* aggressive periodontitis (Fig 6-3) are:

- usually affects patients <30yrs of age (may be older)
- poor serum antibody response to in-

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图 6-2 牙龈雀斑

Fig 6-2 A gingival ephylis (freckle).



图 6-3 牙龈鳞状细胞癌（SCC）

Fig 6-3 A gingival squamous cell carcinoma (SCC) arising within an area of desquamative gingivitis (presumptive diagnosis of erosive lichen planus).

较弱

- 至少3个非第一磨牙或切牙的恒牙出现广泛性的邻间附着丧失。

强调邻间附着丧失的原因是为了与大多数普通非菌斑相关原因所致的附着丧失如刷牙创伤所致的唇面（大多数为颊面）附着丧失相区别。

fecting organisms

- generalised interproximal attachment loss affecting at least 3 permanent teeth that are not 1st molars or incisors.

The reason it is important to specify the term interproximal attachment loss is to differentiate it from one of the most common non-plaque related causes of attachment loss, that of traumatic toothbrushing, which affects facial surfaces (mostly buccal).

1999 年国际专题讨论会分类系统的不足

1999年分类系统（表6-3）具有一些有利的地方，但是在对疾病类型进行分类时许多否定的观点占了上风：

- 发育性或获得性疾病/异常并不是严格的牙周疾病，它们是能够影响所有形式的牙周炎的促进

Critique of the 1999 International Workshop System

The 1999 system (Table 6-3) has some plus points, but a number of negatives having succeeded largely in moving disease categories into different boxes:

- The developmental or acquired conditions/deformities are not strictly periodontal conditions, they are modify-

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因素。

- 由于与"35岁年龄截断"相关的明显分类困难，"侵袭性"这一术语受到欢迎(例如：如果一个患有严重牙周病的患者初次发病时是36岁，属于慢性还是侵袭性呢？)。但如果不考虑年龄更小者将是一个退步，因为在40岁或40岁以上发生新的侵袭性附着丧失者较少见，除非存在明确的相关的局部或系统危险因素。

- 取消"难治性"这一术语是遗憾的，尽管符合逻辑，但是显然仍然存在一小群特殊的个体，他们经过高水平的治疗且菌斑控制水平较高，大多数获得成功的治疗，仍然继续间歇性地遭受牙周组织破坏。也有证据表明这些患者存在共同的潜在危险因素如吸烟和特异性免疫缺陷。

- "坏死性牙周疾病"被再次认识，但NUG是一种局限在牙龈的疾病，根据定义"不"涉及牙周附着装置。尽管应该明确NUG能否发展为NUP，但由于发生NUG时

ing factors that can impact on all forms of periodontitis.

- The term "aggressive" is welcomed owing to the obvious difficulties associated with a 35-years-of-age cut-off (e.g. if the patient is 36 years of age at first presentation, with severe disease, is it chronic or aggressive?). However, the loss of the association with a younger age within the title is a retrograde step, since patients in their 40s and above rarely, if ever, present with new aggressive attachment loss, unless there are recognised local or systemic risk factors associated

- The loss of the term "refractory" is regrettable. Whilst the logic behind this is clear, it is also clear that a small but specific group of individuals exist who, following a high standard of therapy and with high levels of plaque control consistent with successful therapy for the majority, continue to suffer periodontal breakdown at regular intervals. There is also evidence for common underlying risk factors like smoking and specific immune defects in these patients.

- The term "necrotising periodontal diseases" is again understood, but NUG is a condition limited to the gingiva and by definition *not* involving the periodontal attachment apparatus.

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JE没有向根方迁移，所以它们应保持独立。

These terms should remain separate since the JE has not migrated apically in NUG, whilst acknowledgement should be given that NUG may or may not progress to NUP.

- “坏死性口炎”并不属于坏死性牙周疾病，但是如果使用的标准是一致的，它就应该归于这一分类类型。

- The term “necrotising stomatitis” does not appear in the necrotising periodontal diseases list. However, if the criteria applied to this group are to be consistent, it should be added to this category.

- 有许多影响牙龈的疾病没有列出，例如，牙龈雀斑（图 6-4）、牙龈鳞状细胞癌（图 6-5）、病毒疣（图 6-6）等。如果此分类系统要更全面，就应该列出。

- There are many conditions to affect the gingiva that are not listed, e.g. the gingival ephylis [freckle (Fig 6-4)], gingival squamous cell carcinoma (Fig 6-5), viral warts (Fig 6-6), etc. If the system aims to be comprehensive, it should be so.

- 一些所谓的菌斑相关性疾病的“促进因素”并不是促进因素。（例如，维生素 C 缺乏和药物性疾病可能不受菌斑影响）。

- Some of the so-called “modifying factors” for plaque-related disease are not modifying factors (e.g. vitamin C deficiency and drug-induced may be plaque independent).

- 系统性疾病临床表现牙周炎严格来说是一个使用不当的名称，正确的术语应是“系统性疾病的

- Periodontitis as a manifestation of systemic diseases is strictly a misnomer. The correct term should be “pe-

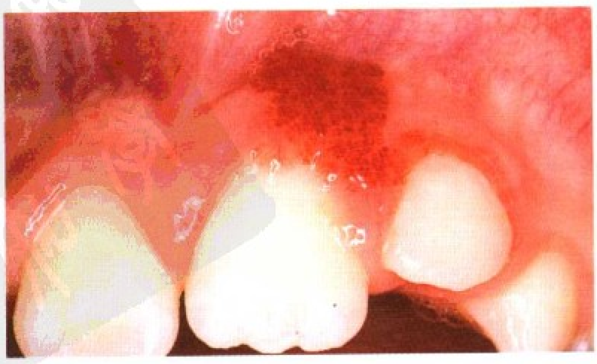


图 6-4　局限性病毒疣

Fig 6-4 A localised viral wart caused by molluscum contageosum.

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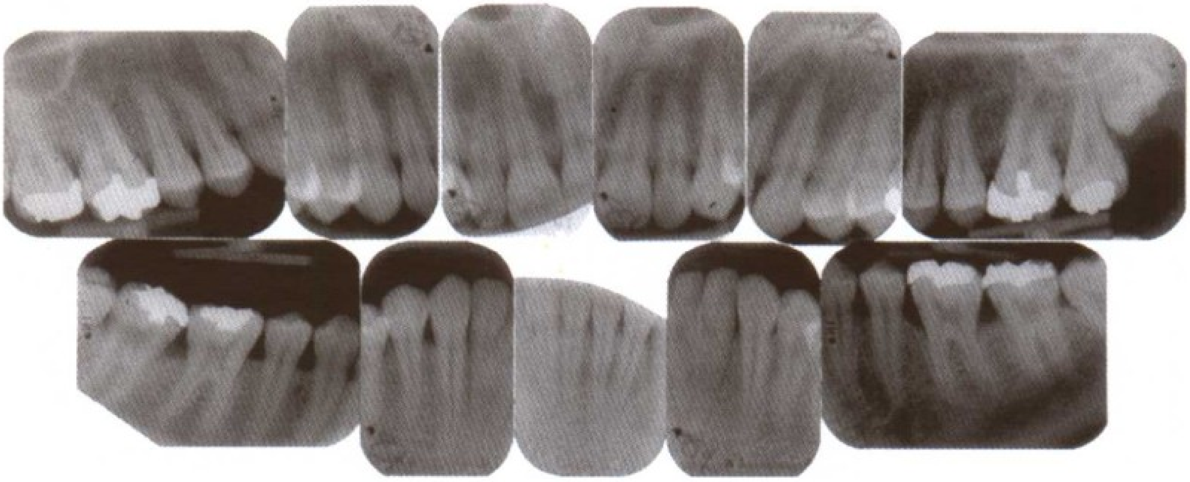


图 6-5　局限性侵袭性牙周炎全口根尖 X 线片

Fig 6-5 Full mouth peri-apical radiographs of localised aggressive periodontitis.

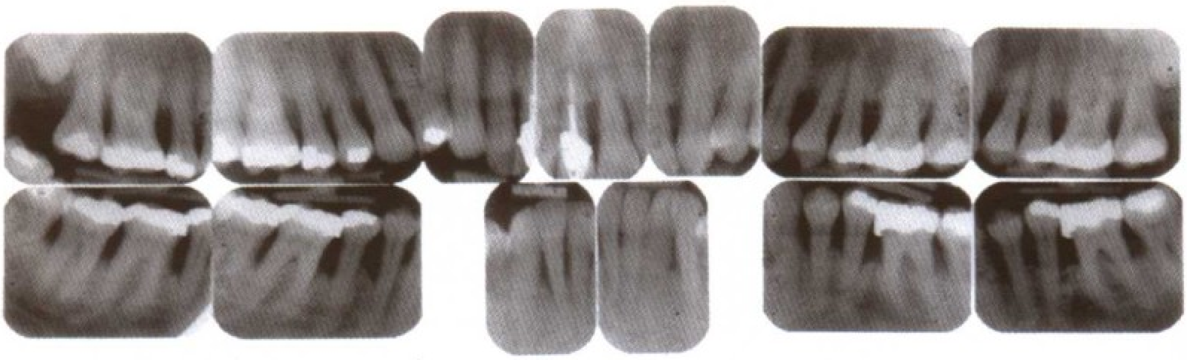


图 6-6　广泛性侵袭性牙周炎全口根尖 X 线片

Fig 6-6 Full mouth peri-apical radiographs of generalised aggressive periodontitis.

牙周表现"。糖尿病不属于这类疾病，因为它能影响任何形式牙周疾病的进展，但如果真如此，它就应该归于Ⅰ部分（发育性或获得性异常和状况）就像作为促进因素的吸烟一样。

• 新分类中主要的问题是取消了

riodontal manifestations of systemic diseases". Diabetes mellitus does not appear in this section, because it can affect the progression of any form of periodontitis, but if this is the case, it should appear under section I (Developmental or acquired deformities and conditions) as should smoking as "modifying factors".

• The main problem with the new clas-

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"局限性青少年牙周炎"（LJP）。LJP 是所有牙周炎形式中最好的典型的定义，具有清晰的临床描述。按作者的意见，在新分类中取消它是令人遗憾的。

sification is the loss of the term "localised juvenile periodontitis" (LJP). LJP is phenotypically the best defined of all forms of periodontitis, with clear clinical descriptors. The loss of the term from the classification system is, in the author's opinion, regrettable.

一个普通牙科治疗的实用性分类

A Practical Classification for General Dental Practice

在试图作出诊断时，临床医师都要努力确定以下问题：

- 疾病史
- 家族史
- 患者的危险因素（如吸烟）
- 健康或疾病
- 龈炎或牙周炎
- 附着丧失的出现、分布和位置
- 真性牙周袋的出现、分布和位置
- 疾病发生时患者的年龄
- 疾病进展的速度
- 基于位点的危险因素的出现、分布和位置
- 骨质破坏的X线检查证据、方式、量和特点
- 溢脓或出血
- 菌斑水平
- 牙齿松动或漂移程度
- 殆因素

When attempting to form a diagnosis, the practitioner generally tries to determine the following questions:

- the history of the condition
- family history
- presence of subject-based risk factors (e.g. smoking)
- health or disease
- gingivitis or periodontitis
- presence, distribution and location of attachment loss
- presence, distribution and location of true pocketing
- age of patient at presentation
- apparent rate of progression
- presence, distribution and location of site-based risk factors
- radiographic evidence, pattern, amount and character of bone loss
- presence of suppuration or bleeding
- plaque levels
- degree of mobility or drifting of teeth
- occlusal factors

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• 疾病是活跃期或处于静止期。（第5章）

实用简化分类见表6-4。

• is the disease active or in remission? (Chapter 5).

A simplified and practical classification system is outlined in Table 6-4.

表6-4 简化分类——根据1999年世界专题研讨会

Table 6-4 Simplified classification - based on world workshop, 1999.

1. 菌斑引起的牙龈疾病	**1. Plaque-induced gingival diseases**
a. 局限性——	a. localised –
Ⅰ. 牙龈脓肿	I. gingival abscess
Ⅱ. 纤维性龈瘤	II. fibrous epulis
Ⅲ. 血管性龈瘤	III. vascular epulis
Ⅳ. 巨细胞性龈瘤	IV. giant cell epulis
周围性	peripheral
中心性	central
b. 广泛性——	b. generalised –
Ⅰ. 无促进因素	I. no modifying factors
Ⅱ. 受性激素影响	II. modified by sex hormones
Ⅲ. 受药物影响	III. modified by medications
Ⅳ. 营养不良影响	IV. modified by malnutrition
2. 非菌斑引起的牙龈疾病	**2. Non plaque-induced gingival diseases**
a. 特异性细菌（如NUG、淋病）	a. specific bacterial (e.g. NUG, gonorrhoea)
b. 特异性病毒（如1度疱疹、手－足－口病）	b. specific viral (e.g. 1° herpes, hand, foot and mouth)
c. 特异性真菌（如LGE）	c. specific fungal (e.g. LGE)
d. 遗传性病损（如HGF）	d. hereditory lesions (e.g. HGF)
e. 系统性疾病的表现	e. manifestations of systemic disease
Ⅰ. 黏膜皮肤疾病（如扁平苔藓／类天疱疮）	I. mucocutaneous disease (e.g. lichen planus/pemphigoid)
Ⅱ. 过敏（如牙膏）	II. allergy (e.g. toothpaste)
Ⅲ. 血液性疾病（如白血病）	III. haematological disease (e.g. leukaemia)
Ⅳ. 肉芽肿疾病（如克罗恩病、结节病）	IV. granulomatous disease (e.g. Crohn's,

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（续　表）

	sarcoidosis)
V．免疫学疾病	V. immunological disease
Ⅵ．其他	VI. other
f．肿瘤（如 SCC，KS）	f. tumours (e.g. SCC, KS)
g．创伤	g. trauma
Ⅰ．烧伤	I. thermal
Ⅱ．化学性	II. chemical
Ⅲ．物理性	III. physical
h．非另外特别说明的	h. not otherwise specified
3．菌斑引起的牙周疾病	**3. Plaque-induced periodontal diseases:**
a．青春前期牙周炎——	a. *prepubertal periodontitis* –
Ⅰ．局限性	I. localised
Ⅱ．广泛性	II. generalised
b．侵袭性牙周炎——	b. aggressive periodontitis –
Ⅰ．局限性（LJP：见教科书）（图 6-2）	I. localised (LJP: see text)
Ⅱ．广泛性（见教科书）（图 6-3）	II. generalised (see text)
Ⅲ．复发性	III. recurrent
c．慢性牙周炎	c. chronic periodontitis –
Ⅰ．局限性（< 30% 位点）	I. localised (<30% sites)
Ⅱ．广泛性（> 30% 位点）	II. generalised (>30% sites)
Ⅲ．复发性	III. recurrent
d．难治性牙周炎	d. refractory periodontitis
4．影响牙周组织的系统疾病	**4. Systemic diseases affecting periodontal tissues:**
a．血液病	a. haematological
b．遗传性疾病	b. genetic diseases
Ⅰ．中性粒细胞减少症	I. neutropenia
Ⅱ．唐氏综合征	II. Down syndrome
Ⅲ．LAD/LLS	III. LAD/LLS
Ⅳ．Chediak-Higashi 综合征	IV. Chediak-Higashi syndrome
V．低磷酸酯酶血症	V. hypophosphatasia

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（续　表）

Ⅵ．组织细胞增多症-X	VI. histeocytosis-X
Ⅶ．Ehlers Danlos 综合征	VII. Ehlers Danlos syndrome
Ⅷ．Papillon-Lefevre 综合征	VIII. Papillon-Lefevre syndrome
c．非另外特别说明的	c. not otherwise specified
5．坏死性牙周炎	**5. Necrotising periodontitis:**
Ⅰ．局限于牙周膜	I. localised to periodontium
Ⅱ．坏死性口炎	II. necrotising stomatitis
6．外侧牙周脓肿	**6. Lateral periodontal abscess**
7．牙周-牙髓联合病变	**7. Periodontal-endodontic lesions**
促进因素	**Modifying factors**
表 6-3“I”中列出	These are listed in Table 6-3 under “I”

注：NUG＝坏死溃疡性龈炎 necrotising ulcerative bingivitis
LGE＝线形牙龈红斑 linear gingival erythema
HGF＝遗传性牙龈纤维瘤病 hereditary gingivo-fibromatosis
SCC＝鳞状细胞瘤 squamous cell carcinoma
KS＝卡波西肉瘤 Kaposi’s sarcoma
LAD＝白细胞黏附功能障碍（异常） leucocyte adhestion defecr
LLS＝惰性白细胞综合征 lazy leucocyte sundrome

理想的分类系统

理想情况下，疾病应该根据病因学或病理学来进行分类。例如，TB 病是结核分枝杆菌单纯感染所致疾病的名称，HIV 是由人体免疫缺陷病毒引起，而心肌梗死就是其字面所述，而不能诊断为“挤压性胸痛”。而对于牙周疾病而言，它们是由多种微生物引起的多种免疫/炎症反应，而每种因素的具体作用在个体之间是不同的。清楚认识三种类型的患者：

The Ideal Classification System

Ideally, diseases should be classified according to their aetiology or pathology. For example, TB is the name given to a mono-infection with *Mycobacterium tuberculosis*, HIV disease is caused by the *human immunodeficiency virus*, and myocardial infarction is what it says and is not diagnosed as “crushing chest pain”. The problem with periodontal diseases is that they are polymicrobial and poly-immune/inflammatory and

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类型Ⅰ——部分个体（8% ONS 1998年联合国个人口腔健康调查）对疾病有系统易感性。

类型Ⅱ——部分个体可由于局部因素的影响而发生疾病。

类型Ⅲ——部分个体对疾病有抵抗力。

将局限性侵袭性牙周炎重新命名为局限性伴放线放线杆菌相关牙周炎会引起大家的兴趣，但是Aa并不总是能够在病变部位分离出，没有疾病发生时也常有Aa的存在（见第2章）。所以，当前牙周疾病应该被看作是一个疾病系列，其结果是附着丧失（图6—7）。在其他方面健康的个体这一模式从本质上将危险（因素）与牙周疾病联系起来，即青春前期牙周炎和系统性疾病／综合征的牙周表现不属于这一模式，因为它们具有清楚明确的遗传基础。

the exact contribution of each factor varies from one individual to the next. What is clear is that there are three categories of patient:

Category I – Some individuals (8% ONS 1998 UK Adult Dental Health Survey) are systemically susceptible to the disease.

Category II – Some individuals can develop the disease due to local factors.

Category III – Some individuals are resistant to the disease.

It would be attractive to be able to rename localised aggressive periodontitis as localised Aa-associated periodontitis, but Aa is not always isolated from diseased sites and is often present when the clinical disease is not (see Chapter 2). For this reason, at present, periodontal diseases should be regarded as a spectrum of disease, the outcome of which is attachment loss (Fig 6-7). This model essentially relates risk to periodontal diseases in otherwise healthy individuals, i.e. pre-pubertal periodontitis and periodontal manifestations of systemic diseases/syndromes fall outside the model, as they have a clearly defined genetic basis.

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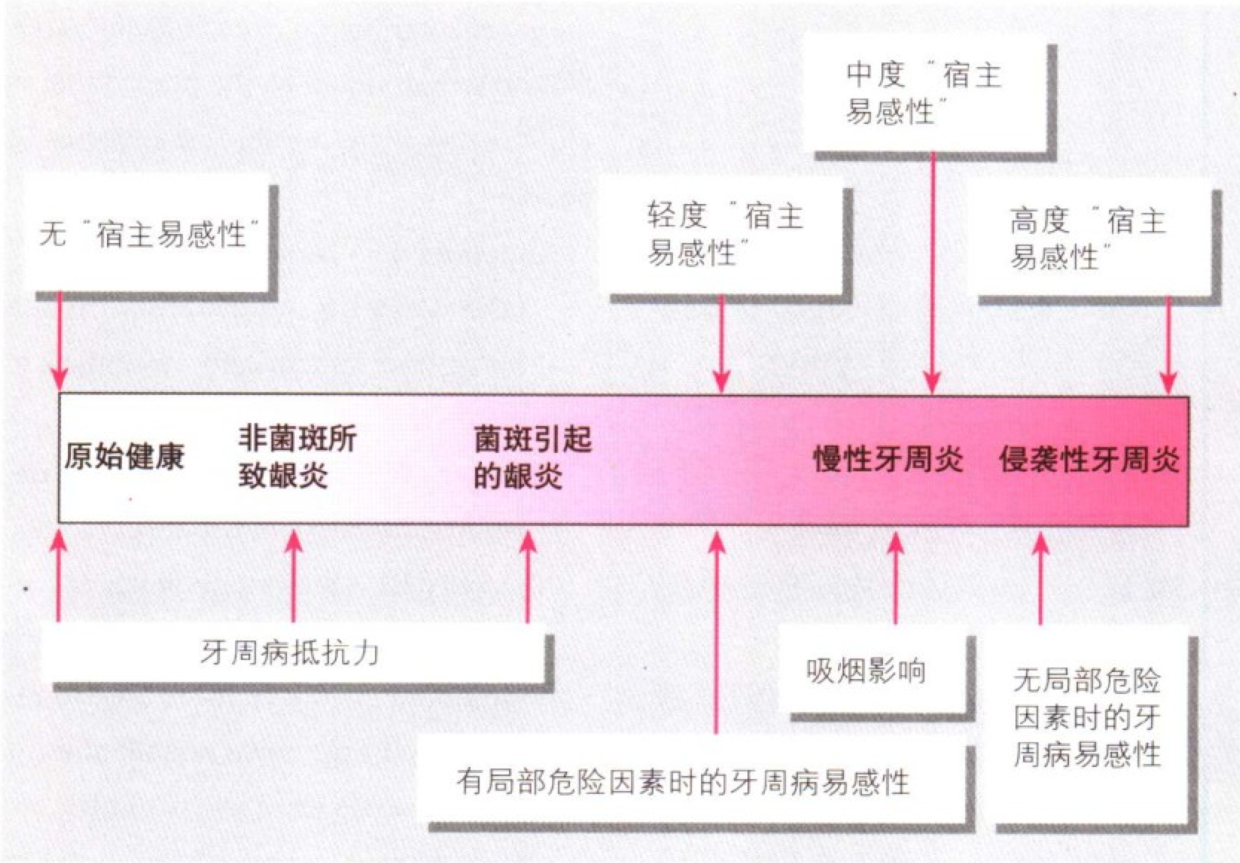


图 6-7　牙周病系列及各种可能影响因素

Fig 6-7 The "spectrum" of periodontal diseases, with proposed markers indicating where various factors may exert an influence.

拓展阅读

Further Reading

1 1999 International Workshop for a Classication of Periodontal Diseases and Conditions. Annals Periodontol, 1999;4:1-112

2 Morris AJ, Steele J, White DA. The oral cleanliness and periodontal health of UK adults in 1998. British Dent J, 2001;191:186-192

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第 7 章 初次就诊——筛选检查

The Initial Consultation – Screening Examination

目　的

本章的目的是给临床医师在评估和检查牙周病患者时提供一个简单的规则系统。

Aim

This chapter aims to provide the practitioner with a simple algorithm for assessing and screening all patients for periodontal diseases.

要　点

学习本章后应该认识到一个准确的、综合全面的病史在牙周病诊断中的重要性。应该能迅速筛查所有患者，识别那些在将来有牙周病危险的患者，同时也应该正确评价推荐的筛查系统的局限性。

Outcome

At the end of this chapter the reader should understand the importance of an accurate and comprehensive history to making a periodontal diagnosis. They should be able to screen all patients rapidly to identify those at risk of periodontal disease in the future, but should also appreciate the limitations of the screening systems recommended.

病　史

当患者初次就诊时，采集全面的病史和检查尤为重要，并在随后的复诊时进行完善更新。如果临床医师要得到正确的诊断并制定合适的治疗计划，这些历史信息是必不可少的，它可被细分为如下几方面。

The History

When a patient first attends for consultation it is important that a thorough history and examination take place and that at subsequent visits these are competently updated. Certain historical information is essential if the clinician is to reach a cor-

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rect diagnosis and develop an appropriate treatment plan. It can be subdivided as follows.

主诉病史

从主诉病史搜集的信息通常非常有用。主要包括：

1. 患者意识到疾病后的持续时间。
2. 判断疾病是否仅仅是局限性的或是否存在系统因素。
3. 对症状的详细描述，包括性质、频率、严重程度、持续时间等。
4. 症状的性质是否随时间发生改变，如数量、频率或严重程度上的改变。
5. 是否有任何加剧或缓解症状的因素。
6. 患者是否曾接受过对此疾病状况的治疗，如果有，治疗的时间和由谁治疗的以及治疗是否有效。
7. 是否存在所患疾病的家族史。

The History of the Complaint

Information gleaned from a complaint history is often extremely useful. The main points to cover are:

1. The length of time the patient has been aware of the problem.
2. Determination of whether the problem is purely localized or whether there is systemic involvement.
3. A detailed description of the symptoms, including nature, frequency, severity, duration, etc.
4. Whether the nature of the symptoms has changed with time, e.g. changes in number, frequency or severity.
5. Whether anything exacerbates or relieves the symptoms.
6. Whether the patient has received treatment for the condition and, if so, when and from whom and whether this therapy was efficacious.
7. Whether there is a family history of the problem.

全身病史

要询问的主要问题已在他处详细列出，此处不需重复。以下是采集这些信息的3个主要原因：

The Medical History

The key questions to be asked are given in detail elsewhere and need not be repeated here. There are, however, three main reasons for collecting this information:

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1. 它可全部或部分解释一种特殊疾病状况出现的原因，并可能解释报告的／观察到的症状／体征的严重程度的原因。
2. 它能提醒临床医师注意系统因素的存在，对此需要在治疗过程中采取特殊的预防措施来保护患者，如抗生素预防。
3. 它能提醒临床医师警惕患者所患的并可能给临床工作人员和以后的患者带来危险的任何疾病，例如乙肝病毒携带者或克雅病（CJD）患者。

1. It may, in whole or in part, explain why a particular condition is seen and may account for the severity of reported/observed symptoms/ signs.
2. It should alert the clinician to the existence of systemic factors for which special precautions will be required to safeguard the patient during treatment, e.g. antibiotic prophylaxis.
3. It should alert the clinician to any disease processes suffered by a patient which may present a risk both to staff in the clinical setting and to subsequent patients, e.g. Hepatitis B carrier or patient with CJD.

社会史

社会史的细节在他处可寻。社会史应包括一个全面的习惯史如吸烟和饮酒，值得注意的是吸烟的年轻女性人数在上升。仔细考虑吸烟的重要性的原因有：

1. 吸烟导致身体许多潜在损伤：
   - 吸烟过程中，尼古丁迅速被血液吸收，其中30%保持自由形式。它们具有高度的脂溶性，故能轻易通过细胞膜，并能特定地影响神经组织包括大脑神经。这可帮助说明心理依赖效

The Social History

Details of what should be covered are available elsewhere. A social history should include a full history of habits such as smoking and alcohol consumption. It is a concern that increased numbers of young women in particular are smoking. It is important to consider tobacco smoking carefully because:

1. Tobacco smoking causes many potentially damaging changes in the body:
   - During smoking, nicotine is rapidly absorbed into the blood stream where 30% remains in free form. It is highly lipid-soluble and thus gains easy passage across cell membranes, where it appears particularly to affect neu-

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应的原因。

• 尼古丁加快心率和增加了心脏排血量，升高血压。它也能直接作用于血管，诱发血管收缩。

2. 吸烟也可引起潜在的口腔损害：

• 长期来自香烟对口腔黏膜的温度和化学刺激可导致口腔黏膜的改变，从相对良性的状态如吸烟角化病、烟草性口炎以及潜在的更恶性状况如黏膜白斑到症状明显的癌。吸烟产生的主要致癌物包括多环芳香烃和N-亚硝基复合物。

• 烟草的烟具有选择性的杀菌作用，能引起口腔菌群发生变化，导致易于感染念珠菌病。

• 牙齿和软组织上的外来色斑是由于焦油产物的沉积所致。牙齿上的色斑由于表面粗糙促进了菌斑积聚，在“危险患者”，可促进牙周疾病的发生。

ral tissue including the brain. This may help account for the psychological dependence effects.

• Nicotine increases heart rate, cardiac output and blood pressure. It also acts directly upon blood vessels inducing vasoconstriction.

2. Tobacco smoking also causes potentially damaging oral changes:

• Prolonged thermal and chemical irritation of oral mucosa from smoke may result in changes in the oral mucosa ranging from relatively benign conditions such as "smoker's keratosis" and "nicotinus stomatitis" and more potentially sinister conditions such as leukoplakia through to frank carcinoma. The principal carcinogens within tobacco smoke include polycyclic aromatic hydrocarbons and N-nitroso compounds.

• Tobacco smoke is selectively bactericidal and causes changes in the oral microflora, resulting in a predisposition to candidosis.

• Extrinsic staining of the teeth and soft tissues occurs due to deposition of tar products. Staining of the teeth encourages plaque accumulation due to the rough surface of the stain, which may, in "risk patients", result in increased periodontal disease experience.

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• 吸烟在NUG的病因学中是高度关联的。
• 吸烟者比不吸烟者产生更多的牙石。烟中的刺激物导致唾液流速加快。当唾液从腮腺流出，特别是流速加快时，产生的唾液具有更高的pH值和升高的钙浓度，它能使磷酸钙的沉淀增加。牙石本身相对来说无致病作用，但却提供了一个菌斑积聚的粗糙面，使易感患者易于感染牙周疾病。
• 流行病学调查证据表明吸烟者比不吸烟者具有更高的菌斑指数。这并不能认为是吸烟的直接效果，但能确信与不良菌斑控制有关。
• 越来越多的证据表明吸烟人群牙周疾病的发生率、流行情况和严重程度均比不吸烟人群高（见第 4 章）。
• 吸入尼古丁导致牙龈血管收缩，掩盖了牙周袋探诊出血(BOP)这一重要体征。所以对吸烟人群应仔细检查，否则可能低估牙周疾病的活动性。

• Smoking is heavily implicated in the aetiology of NUG.
• Smokers produce more calculus than non-smokers. It is thought that irritation from the smoke results in an increased salivary flow rate. When saliva flow from the parotid gland, in particular, increases, the saliva produced has a higher pH and a raised concentration of calcium, resulting in increased precipitation of calcium phosphate. Calculus *per se* is relatively inert but it does have a rough surface which is plaque retentive, predisposing to periodontal problems in susceptible patients.
• Epidemiological evidence suggests that smokers have higher plaque scores than non-smokers. This is not thought to be a direct effect of the smoking but is believed to be related to poorer plaque control.
• There is increasing evidence demonstrating that the incidence, prevalence and severity of periodontal disease seen in smokers is greater than in non-smokers (see Chapter 4).
• Vasoconstriction within the gingivae resulting from nicotine inhalation masks the important sign of BOP from the base of the pocket. Smokers must thus be examined carefully, otherwise the activity of periodontal

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disease may be underestimated.

- 血管收缩也可降低GCF的流量。在第3章已讨论过，GCF中含有许多宿主防御产物，它们的减少是潜在的损害。

- Vasoconstriction also results in reduced GCF flow. As discussed in Chapter 3, this contains many host-defence products, whose reduction is potentially damaging.

- 已经证实来自于吸烟人群龈沟内的PMNLs的趋化性、吞噬作用和迁移速度降低（见第3章）。免疫-炎症细胞功能降低与牙周炎的侵袭方式有关。

- It has been demonstrated that PMNLs derived from the gingival crevice in smokers show reduced chemotaxis, phagocytosis and migration rate (see Chapter 3). Reduced immune-inflammatory cell function is associated with aggressive forms of periodontitis.

- 吸烟降低了手术和非手术牙周治疗的成功率。吸烟人群中尼古丁被吸附在牙面上并能导致成纤维细胞定向障碍。

- Smoking reduces the success of both surgical and non-surgical periodontal therapy. Nicotine is adsorbed onto the root surface in smokers and can result in fibroblast disorientation.

3. 口腔医师在劝说戒烟和帮助那些希望减少或希望停止吸烟的患者达到目的过程中起着重要作用。关于如何正确地戒烟有大量的有效的信息资料，但必须让患者知道戒烟将获得口腔健康。

3. The dental team can play an important role in smoking cessation counselling and in helping patients who wish to reduce or stop smoking to achieve this. A great deal of information is available about how correctly to approach smoking cessation, but it is essential that patients know that quitting smoking will produce an oral health gain.

当成功的可能性降低时，是否应该对吸烟人群拒绝使用那些有效的牙体/牙周治疗方法是一个感情问题。植入种植体和引导组织再生手术常不在

The question of whether certain forms of dental/periodontal therapy should be denied to smokers on the grounds that the likelihood of success is reduced is an emotive

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吸烟人群进行，而传统牙周手术则可能使用，当一种治疗对临床医师来说是需要技术和耗时的，且对患者来说是昂贵的时候，双方都希望获得最大的成功机会。

issue. Implant placement and guided tissue regeneration are often *not* offered to smokers, while conventional periodontal surgery may be. When a treatment is technically demanding and time-consuming for the clinician and costly for the patient, both may wish to maximise the chance of a successful outcome.

因为慢性酒精中毒对肝功能和血凝的影响，所以从牙周观点出发，饮酒的作用也是重要的。

Alcohol consumption is also important from a periodontal standpoint owing to the effects of chronic alcoholism on liver function and hence blood clotting.

检　查

检查从口外开始，正常要求包括 TMJ 功能、口腔颌面肌肉和局部淋巴结的检查，接着是全面的口内检查，总的来说包括口内软组织和硬组织。为此设计了一套临床检查工具如图 7-1 所示，尽管每位医师都愿意拥有他们自己更喜欢的工具。

The Examination

The examination begins extraorally and normally includes examination and, as required, testing of the function of the temporomandibular joints (TMJ), the orofacial musculature and regional lymph nodes. This is followed by a general intra-oral examination designed to look at the soft and hard tissues within the mouth as a whole. An examination kit designed to achieve this



图 7-1　临床检查基本器械

Fig 7-1 Photograph illustrating the basic instruments required to perform a clinical examination.

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检查应考虑的内容包括：

- 美学。患者是否对此满意？如果不满意，可能极大地影响最终的治疗计划。
- 龈色。尽管由于个体差异比较大，很难将之归于一种特定的颜色，但习惯性地将正常牙龈描述为"粉红色"（见第5章）。有的炎症区域明显发红，很易被识别。
- 牙龈外形。牙龈外形较易观察，可能呈现肿胀或退缩。急性炎症可引起牙龈肿胀，此时牙龈发红、水肿，牙龈肿胀也可由慢性炎症所致，此时常有更多的纤维变性。在两种情况中菌斑都是病理学的始发因素，其他因素如激素水平的改变能加剧菌斑引起的反应。许多药物都有患者不希望出现的牙龈增生的副作用，尽管不是所有患者均受到影响且受影响的程度差别很大。这类药物包括：

 - 苯妥英（如大仑丁®），用于治疗癫痫。

is shown in Fig 7-1, though each clinician will have their own preferred instruments.

The examination should include consideration of:

- Aesthetics. Is the patient content with this? If not, this may significantly affect the ultimate treatment plan.
- Gingival colour. Normal gingivae are conventionally described as being "pink", though within the wide limits of individual variation it can be difficult to ascribe a particular colour (see Chapter 5). Some inflamed areas are markedly red and are easily identified.
- Gingival contour. This is easier to see and there may be either swelling or recession/shrinkage. Swelling may be due to acute inflammation, in which case the gingivae are red and oedematous, or it may be caused by chronic inflammation, in which case there is often much more fibrosis. In both situations plaque is the initiator of the pathology and the response to it can be exacerbated by other factors such as hormonal change. A number of medications have the undesired side-effect of gingival overgrowth, though not all patients are affected and the degree of involvement may vary greatly. The drugs in question include:

 - phenytoin (e.g. Dilantin®), used to treat epilepsy

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– 钙通道阻断药（如氨氯地平），用于治疗高血压和心绞痛。
– 环孢素，用于移植手术后预防器官排斥反应。

- 牙龈退缩。应该注意到，在受到影响的位置可能存在或可能不存在炎症和（或）牙周袋。有时牙龈退缩是局限性的，但也可能是广泛性的。牙龈退缩有许多不同的原因，包括过去的牙周病以及在相对缺乏牙槽骨的部位使用创伤性的刷牙方法。牙龈退缩的地方不可避免地出现牙根暴露，并可导致根面龋和（或）牙本质过敏。如果根分叉部位受累，可能会有牙髓活力丧失（见第 8 章）。
- 溢脓。这可能是患者的主诉或被患者描述为有臭味。临床上可能表现为瘘道，在探查一个位点后脓液可从龈缘下溢出，或轻压牙龈时溢出。应该找出溢脓的原因，因为仅仅对症治疗（例如服用抗生素）在长期的治疗中将是失败的。
- 修复体的质量和完善。这在评估牙周病和设计治疗计划时非常

- calcium channel blockers (e.g. amlodipine), used to treat hypertension and angina
- ciclosporin, used post-transplantation to prevent organ rejection.

- Recession – This should be noted, and there may or may not be inflammation and/or pocketing at affected sites. Sometimes the problem is localised, but it may also be generalised. There are a number of different causes of recession, including past periodontal disease and a traumatic toothbrushing technique at sites with a relative lack of bone. Affected sites inevitably have root exposure, which may result in root caries and/or dentine hypersensitivity. If furcation areas are thus affected, loss of vitality may occur (see Chapter 8).
- Suppuration. The patient may report this directly or a bad taste may be described. Clinically it may be seen as a sinus, it may exude from beneath the gingival margin after a site has been probed, or it may be elicited by gentle massaging of the gingivae. The cause should always be identified, as treating the symptoms only (e.g. by prescribing antimicrobials) will not be successful in the medium to long term and is bad practice.
- Quality of restorations and their integrity. This is extremely significant

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重要。简言之，有缺陷的修复体边缘起着菌斑携带物的作用，而且龈下部分非常难以清洁。在易感患者积聚的菌斑将导致更多的牙周疾病，在牙周炎易感患者，塑料义齿、冠、桥和种植体等正、反两面有缺陷的修复体边缘应该得以纠正，这是极其重要的。也应该注意龈下边缘并仔细探查。

when the periodontal condition is being assessed and when a treatment plan is to be devised. In short, defective margins act as plaque traps, and if subgingival are extremely difficult to clean. In susceptible patients the accumulated plaque will result in more periodontal disease and it is vital that both positive and negative defective margins on plastic restorations, crowns, bridges, implants, etc. are corrected in periodontitis-susceptible patients. Subgingival margins should also be noted and carefully probed.

• 由于上述同样原因，也应检查口内活动矫正器。这包括正畸矫正器和修复矫正器。

• Removable appliances in the mouth should be examined for the same reasons as above. This would include orthodontic and prosthetic appliances.

• 龋齿包括根面龋，如果存在这一情况，应进行正规的饮食记录并开始执行一个全面的预防计划。

• Caries if present should be noted including root caries. If there is a particular problem in this regard, a formal diet history should be elicited and a full preventive programme commenced.

• 在检查潜在的牙周炎促进因素时，殆关系检查是必不可少的（见第 6 章）。可能存在牙齿磨损，有记录的TMJ疾病（包括疼痛、强直、牙关紧闭、偏斜的开口路径），牙齿移位，逐渐增加的牙齿松动度，或创伤性覆殆。临床上殆关系检查应包括所有的咬合运动，必要时也可使用研

• Occlusal examination is essential, to check for modifying factors of an underlying periodontitis (see Chapter 6). There may be evidence of tooth wear, reported TMJ problems (including pain, stiffness, trismus, deflected opening), tooth migration, increasing tooth mobility, or the presence of a traumatic overbite. The occlusion

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究模型。如果治疗计划很复杂，同时包括重要的修复工作，则必须进行殆关系评估。

should be checked clinically in all excursions and study models may also be necessary. If it is envisaged that the treatment plan will be complex, involving significant restorative work, occlusal assessment is mandatory.

显然，探查到的任何异常都需要进一步的调查，在任何情况下所有的结果包括阴性结果都应该仔细地记录下来并清楚地签名和注明日期。

Clearly, any abnormalities detected require further investigation and in any event all results, including those of negative findings should be carefully recorded and the notes legibly signed and dated.

对牙周组织，以下是一个合理的出发点。如果患者对你来说是一个新患者或在近一年内未接诊过的患者，使用英国牙周病学学会推荐的基础牙周检查（BPE）是切合实际的。这是一个快速筛选系统，已被用来帮助识别有重要牙周疾病的"危险患者"，而这些危险患者需要更详细的牙周检查。它不是对易感患者详细检查的快速替代方法。

Moving to the periodontal tissues, the following is a reasonable starting point. If the patient is new to you or has not been seen within the last year it is sensible to use the Basic Periodontal Examination (BPE) as recommended by the British Society of Periodontology (BSP). This is a rapid screening system which has been developed to aid the identification of "risk patients" with a significant periodontal problem, who therefore require a more detailed periodontal examination. It is not a quick alternative to a detailed examination in susceptible patients.

基础牙周检查

探　诊

探诊检查时需要一种特殊探针——WHO探针。这种探针有多种变化。图 7-2 和 7-3 显示其重要的标志和尺寸。关键的要素包括：

The BPE

The Probe

A special probe is required called the WHO (World Health Organization) probe. There are several variations upon the theme but the significant markings and measurements are as shown in Figs 7-2 and 7-3.

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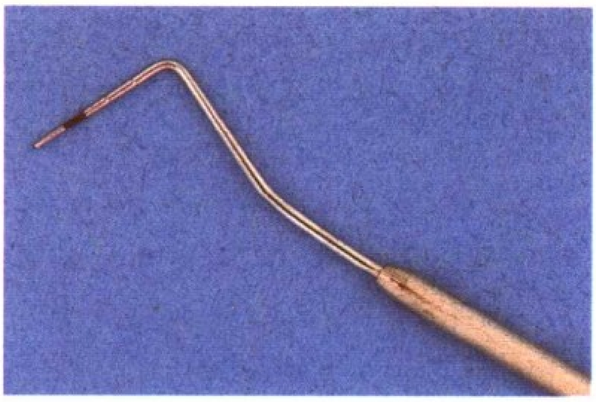


图 7-2 WHO-E 型探针

Fig 7-2 Photograph illustrating the basic points of a WHO-E (epidemio-logical)-type probe.

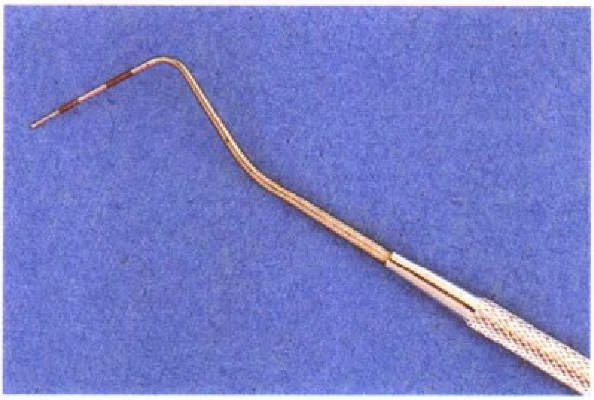


图 7-3 WHO-C 型探针

Fig 7-3 Photograph illustrating the basic points of a WHO-C (clinical)-type probe.

- 直径为 0.5 mm 的球状末端。
- 彩色带从3.5 mm延伸到5.5 mm（WHO-E 型探针）。
- 在WHO-C型探针上有第二条带，从8.5 mm到11.5 mm（WHO-C-型探针）。
- 探诊力量不应该超过 0.2～0.25 N（20～25 g）。

The key elements include:

- a ball-end of 0.5mm diameter
- a coloured band extending from 3.5 to 5.5mm (WHO-E (epidemiolog-ical)-type probe)
- a second band is present in certain types of probe (WHO-C (clinical)-type probe) from 8.5 to 11.5mm
- the target probing force should not exceed 0.2-0.25N (20-25g weight).

牙列分区

牙列分为如下六个区（FDI 标志法）：

Division of the dentition

The dentition is divided into sextants as below (FDI notation):

17 – 14	13 – 23	24 – 27
47 – 44	43 – 33	34 – 37

如果所在区没有牙齿则在表中以叉记录（图 7-4）。如果一个区只有一颗牙，则该区像先前一样记作无牙，该孤立牙包括在邻近的区内。

If there are no teeth in a given sextant this is recorded as a cross in the chart (Fig 7-4). If only one tooth is present in a sextant, the sextant is recorded as having

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姓　名：A. N. Other　　　　日　期：1 November 2002

×	4	×
×	1	×

×代表该区域无牙齿

图 7-4　基础牙周检查图表
Fig 7-4 Diagram of a BPE chart showing a cross in the sextants which have one or no teeth present.

no teeth present as previous, and the single tooth is included in the adjacent sextant.

怎样进行探诊？

将牙周探针尖轻轻地放在牙周袋底，对照颜色刻度读出插入深度。用牙周探针在每个区内环绕牙龈边缘轻轻地"爬行"来方便地探查整个牙周袋范围。每颗牙至少应该检查六个点：近中颊、颊和远中颊和对应的舌侧位点。每个区只记录一个评分/编码，即该区任何位点最高的记录值。

How?

The probe tip is gently placed to the base of the pocket and the depth of insertion read against the colour coding. The total extent of the pocket should be explored conveniently by gently "walking" the probe around the entire gingival margin within each sextant. At least six points on each tooth should be examined: mesio-buccal, mid-buccal and disto-buccal and the corresponding lingual sites. For each sextant only one score/code is recorded and that is the highest encountered anywhere within it.

评分／编码系统

该系统最好用图中的方式进行描述（图 7-5 和 7-6）。文字描述如下：

"0"：在该区最深的牙周袋内探针的有色带全部可见。牙龈组织健康，轻

The scoring/coding system

This is best illustrated pictorially (Figs 7-5 and 7-6). In words:

Code 0 The coloured band on the probe is completely visible in the deepest pocket

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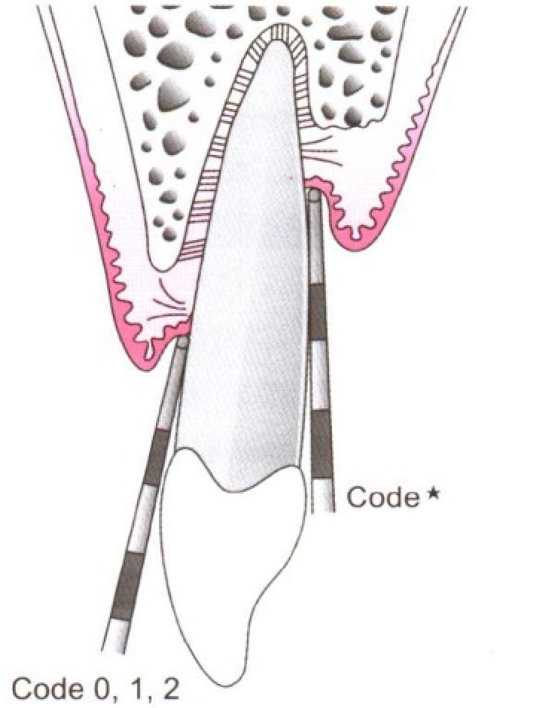


图 7-5 BPE 编码系统（0，1，2+，★）

Fig 7-5 Diagram illustrating the BPE coding system in codes 0, 1,2 + ★.

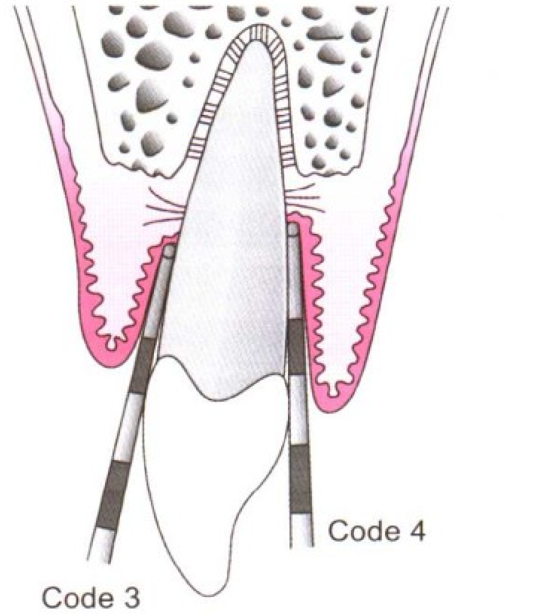


图 7-6 BPE 编码系统（3，4）

Fig 7-6 Diagram illustrating the BPE coding system in codes 3 and 4.

探无出血。

"1"：在该区最深的牙周袋内探针的有色带全部可见。无牙石和有缺陷的菌斑滞留的修复体边缘。轻探出血。

"2"：探测该区最深的牙周袋时探针的有色带全部可见。或者有牙石（龈上或龈下）存在，或者有滞留菌斑的/有缺陷的修复体边缘。

"3"：探测该区最深的牙周袋时探

in the sextant. The gingival tissues are healthy, displaying no bleeding after gentle probing.

Code 1 The coloured band on the probe is completely visible in the deepest pocket in the sextant. There is no calculus and no plaque-retentive defective restoration margins are present. There is bleeding after gentle probing.

Code 2 The coloured band on the probe is completely visible in the deepest pocket in the sextant. Either, calculus (supragingival or subgingival) is present or there is a plaque-retentive/defective restoration margin.

Code 3 The coloured band on the probe

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针的有色带仅部分可见，表示牙周袋深度为 3.5～5.5 mm。

"4"：探测该区最深的牙周袋时，探针有色带完全隐藏，表示牙周袋深度≥ 6 mm。

"★" 表示两种特殊情形：

- 附着丧失≥ 7 mm 或
- 累及根分叉

记录信息

可通过框图表来完成（图 7-7）

根据评分治疗

根据记录的评分 / 编码，该系统提出以下治疗：

"0"：不需治疗。

"1"：口腔卫生宣教（OHI）。

"2"：口腔卫生宣教加上去除牙石和（或）纠正菌斑滞留的修复体边缘。

is only partly visible in the deepest pocket in the sextant, indicating a PPD of 3.5-5.5mm.

Code 4 The coloured band on the probe is hidden in the deepest pocket in the sextant, indicating a pocket depth of ≥ 6mm.

Code★ This denotes two special features:

- loss of attachment of ≥ 7mm or
- furcation involvement.

Recording the information

This is done using a box chart (Fig 7-7).

Management according to score

The system suggests the following managements according to the score/code recorded:

Code 0: No treatment required

Code 1: Oral hygiene instruction (OHI)

Code 2: OHI plus calculus removal and/or correction of plaque-retentive restorative margins.

姓　名： A. N. Other　　　　**日　期：** 1 November 2002

★	4	3
2	1	×

×代表该区域无牙齿

图 7-7　完成的 BPE 图表

Fig 7-7　Diagram illustrating a completed BPE chart.

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所有区 BPE 评分为 0、1 或 2 的患者一年后均应再次进行筛选检查。

Patients with BPE scores of 0, 1 or 2 in all sextants should be screened again after one year.

"3"：口腔卫生宣教加上根据需要进行龈上洁治，纠正修复体边缘和根面清创（RSD），在治疗的开始和结束时均应记录菌斑和出血评分。治疗结束时评分为 3 时在区内的应记录牙周袋图表（见第 8 章）。另外，在这些区内应该在不超过一年的间隔时间再次记录菌斑、出血和牙周袋图表，同时对其他区进行 BPE 筛选检查。

Code 3: OHI plus scaling, restoration margin correction and root surface debridement (RSD), as required. Plaque and bleeding scores should be recorded at the start and finish of treatment. Pocket charts (see Chapter 8) should be recorded in sextants scoring code 3 at the finish of treatment. In addition, such sextants should have plaque, bleeding and pocket charts re-recorded at intervals of not more than one year along with BPE screening of the other sextants.

"4" 或 "★"：口腔卫生宣教加上根据需要进行龈上洁治，修复体边缘修正和RSD，稍后可能需要进行牙周手术治疗，且应该考虑求助于牙周病学专家。治疗开始时应记录菌斑、出血和详细牙周袋图表，同时记录牙龈退缩，根分叉病变和任何其他相关的临床细节。对有根分叉病变或附着丧失≥7mm的牙齿应进行口内X射线检查（见第 8 章）。治疗后应该进行全面的再检查来评估对治疗的反应和进一步治疗的潜在要求，它可能包括手术治疗。

Code 4 or Code★: OHI plus scaling, margin correction and RSD as required. Surgery may be required later and referral to a specialist in periodontology should be considered. Plaque, bleeding and detailed pocket charts together with recordings of gingival recession, furcation involvement and any other relevant clinical details should be recorded at the start of treatment. Individual intra-oral radiographs of teeth which show furcation involvement or loss of attachment of ≥ 7mm should be taken (see Chapter 8). There should be a full re-examination after treatment to assess both the response to therapy and the subsequent potential requirement for further treatment, which may include surgery.

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基础牙周检查的优点和缺点

同所有筛选测试一样，BPE有优点和缺点，如果要正确地应用并最大的受益，有必要对它们进行认识。

BPE **的优点**

主要有七点：

1. 它是一个简单而迅速的筛查系统，能帮助识别那些患有严重牙周疾病的个体，他们还需要更加详细的牙周检查。然而BPE并不是一条捷径，也不能替代对该类病例的详细检查。从公共健康的观点出发，对严重感染者的识别能够有助于将稀有资源留给那些最需要的人。
2. 本系统在国际上得到很好的认可，能够帮助我们跨越地域的界限进行信息交流。
3. 检查既迅速又容易完成。
4. 检查需要的惟一器械是一支合适的牙周探针，既不昂贵又易获取。
5. 系统以一种容易交流的形式概括牙周疾病。

What are the Advantages and Disadvantages of the BPE?

As with all screening tests the BPE has benefits and disadvantages, and if it is to be used both correctly and to best advantage, knowledge of each is essential.

Advantages of the BPE

There are seven main ones:

1. It is a simple and rapid screening system which aids the identification of those individuals with significant periodontal disease who then require a much more detailed periodontal examination. However, the BPE is neither a short cut nor an alternative to a detailed examination in such cases. From a public health standpoint, identification of significantly affected persons should aid the targeting of scarce resources at those in most need.
2. The system is well recognised internationally, which aids communication of information across geographical boundaries.
3. The examination is both rapid and easy to complete.
4. The only equipment required is an appropriate probe and this is both readily available and inexpensive.
5. The system summarises the periodontal condition in a readily communi-

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cable form.

6. 总之，系统对适合指定编码的治疗给出了指征，并对部分编码提出应该考虑何种治疗安排。

6. The system, as a whole, gives an indication of treatment appropriate to the codes assigned and suggests in some codes whether referral should be considered.

7. 也许由于上述因素的缘故，关于BPE最好的一点是它鼓励和促进了全科治疗时牙周组织的检查。这对临床医师和患者都是很重要的。在今天，未诊断的牙周疾病是牙科诉讼案件中一个主要原因。

7. Perhaps because of a combination of the above factors, one of the best things about the BPE is that it encourages examination of the periodontal tissues in general practice. This is important for both clinicians and patients. Certainly in the current day, undiagnosed periodontal disease is one of the major causes of litigation in dentistry.

BPE 的缺点

有各种各样的缺点，包括：

1. *在分区内缺乏详细说明。*这是任何分级编码系统的必然结果，这一编码系统只需记录分区内最高指数。BPE没有详细的受影响的位点数目、也没有感染严重程度的详细说明，这会引起混淆。例如，一个区内有一个6 mm牙周袋时记录为4，而一个区内广泛地存在5 mm牙周袋时则会记录为3。仅看记录的编码会认为前者的牙周疾病要比后者严重。所以结果的细致解释尤其重要。

Disadvantages of the BPE

These are varied and include:

1. *Each of detail within sextants.* This is an inevitable consequence of any hierarchical coding system, which requires only the highest score in any sextant to be recorded. The BPE provides no detail about either the number of affected sites or the severity of involvement, which can lead to confusion. For example, a sextant with one 6mm pocket would be assigned a code 4 while a sextant with generalised 5mm pockets would be assigned a code 3. Looking only at the codes one would assume that the periodontal condition was worse in the former rather

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than the latter case. Careful interpretation of results is thus vital.

2. *所有的编码中缺乏疾病活跃性信息。*这是多面分级编码系统的结果。牙周袋探诊出血这一重要体征只在两个编码中提到，编码"0"表示无探诊出血，编码"1"表示探诊出血。一旦记录编码为"2"或更高时，BOP则与该指数无关。很多人认为编码"2、3、4和★"表示有这一体征，但事实并非如此，所以这一系统在后面的编码中没有给出潜在的疾病活跃性的指征。

2. *Lack of information on disease activity in all codes.* This is a consequence of a multi-faceted hierarchical coding system. The important sign of bleeding on probing from the base of the pocket is specifically referred to in only two codes – code 0 where it is not present and code 1 where it is. Once code 2 or higher is recorded, BOP becomes irrelevant to the index. Many people assume that this sign is present and part of codes 2,3,4 and ★ but this is not so – thus the system gives no indication of potential disease activity in these latter codes.

3. *不能提供附着丧失（LOA）的信息。*这是该编码系统的固有结果，因为所有的编码都是仅依赖牙周袋探诊深度进行指定。只有编码"★"例外，它表示涉及根分叉病变或LOA ≥ 7 mm。缺乏LOA的详细情况是一个严重缺点，因为这一重要的测量值对疾病经历提供了极其重要的信息。

3. *Inability to provide information on loss of attachment* (LOA). This is an inherent consequence of the system as in all codes bar one assignation of code is based purely upon probing pocket depth. The exception is code ★ which implies either the presence of furcation involvement or LOA of ≥ 7mm. Lack of detail about LOA is a serious disadvantage as this important measurement gives vital information about disease experience.

4. *不能区分真性和假性牙周袋。*这是该系统仅依靠牙周袋深度测量的必然结果，这也使治疗计划的制定变得困难。例如，对

4. *Inability to differentiate between true and false pockets.* This is a consequence again of having a system based almost exclusively on pocket depth

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一个5 mm BOP（+）的真性牙周袋，治疗应该选择根面清创（RSD），而如果是假性牙周袋，RSD则完全不合适，因为在牙冠部不会出现新附着。

5. *缺乏根分叉病变的详细情况。*编码“★”表示在一个区内可能存在根分叉病变，但并没有给出足够的信息。理想情况应该知道哪颗牙涉及根分叉病变，哪个（哪些）根分叉受损，以及根分叉病变的严重程度（见第8章）。

6. *BPE不能应用在年轻人群。*因为年轻患者的全部临床牙冠还没有完全萌出，龈缘常常位于ACJ（amelo-cemental junction）的冠方，应用依靠牙周袋深度编码系统的BPE将导致过高估记编码值。

7. *BPE不能用于监控疾病。*尽管这样的尝试是诱人的，但是由于在分区内缺乏详细的有价值的信息，BPE不能用来监控疾病。企图用BPE编码进行比较是毫

measurements. This makes treatment planning difficult. For example, the treatment of choice for a 5mm *true pocket*, which exhibited BOP from the base of the pocket, would be to root plane the site (perform RSD). If the pocket was false, however, RSD would be entirely inappropriate, as new attachment onto the tooth crown could never occur.

5. *Lack of detail about furcation involvement.* Code★ implies that furcation involvement may be present in a sextant but it does not give sufficient information. Ideally one should know which tooth is involved, which furcation) is/are involved and what the severity of the involvement is (see Chapter 8).

6. *BPE cannot be used for young individuals.* The gingival margin is often positioned coronal to the ACJ in young patients, as full clinical crown length maturation has not occurred. Using the BPE with its coding system based on pocket depth would thus result in a tendency to overestimate the code assigned.

7. *BPE cannot be used to monitor disease.* Although it is tempting to try, the BPE cannot be used to monitor disease due to the lack of detailed information available within sextants. At-

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无意义的。

8. *需要一种特殊探针*。尽管容易获得，但需要一定成本。

tempts at comparison using BPE codes only would be inherently meaningless.

8. *A special probe is required.* Although these are readily available, there is a capital cost involved in procuring them.

基础牙周检查是一个好方法吗？

除了应该被理解的局限性和应该仔细解释的评分系统，它确实提供了一种简单快捷的评估方法来判断是否存在重要的牙周疾病，如果存在则建议继续对感染区进行详细的检查。为了判断是否在将来有附着丧失的危险，没有理由不检查每一个患者的牙周状况。由于所需费用低、时间少，BPE是一种实用而灵敏的方法。

Is the BPE a Good Thing?

The million-dollar question! Basically, the answer is yes, provided the limitations are understood and the scores carefully interpreted. It does provide a quick and simple means of assessing whether significant periodontal disease is present in a mouth, and if so recommends detailed examination of the affected sextants. There is no excuse for not examining the periodontal condition of every patient, to determine those at risk of future attachment loss. For a minimal financial outlay and minimal time, BPE is practical and sensible.

拓展阅读
Futher Reading

Lindhe J, Karring T, Lang NP(Eds.) Clinical Periodontology and Implant Dentistry. 3rd ed. Copenhagen: Munksgaard, 1998

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第8章 详细的临床牙周检查

The Detailed Clinical Periodontal Examination

目 的

本章概括了牙周炎易感患者详细牙周检查的主要内容。

Aim

The purpose of this chapter is to outline the key stages involved in examining in detail patients identified as being susceptible to periodontitis.

要 点

希望读完这部分后，临床医师能理解推荐的个体测量方法的原理，进而能执行、记录和解释这种评估方法，并制定出正确的治疗计划。

Outcome

It is anticipated that having read this section, practitioners will understand the rationale for the individual measures recommended, and that they will be in a position to perform, record and interpret such assessments and produce appropriate treatment plans.

临床牙周检查

完成口外、常规口内和牙周筛选检查后，就应该清楚是否需要详细的牙周检查。如果需要，则不可避免地需要大量时间，很遗憾没有其他选择。由于疾病的位点特异性，所有的位点都需要非常仔细地进行检查（见第5章）。总的来说，对有限的几个位点的检查

The Clinical Periodontal Examination

Having completed the extra-oral, general intra-oral and periodontal screening examinations, it should be apparent whether a detailed periodontal examination is necessary. If so, this will inevitably take a significant amount of time, but there are unfortunately no current alternatives. All

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无法对口腔作出任何诊断假设。

详细的临床牙周检查包括五个独立的部分，且总是先于任何放射检查或其他特殊检测。通过从中获得的信息，临床医师能够在逻辑基础上决定是否需要另外的检测以及需要什么样的检测。这能保证只进行必要的检测。这五个部分是：

- BOP（+）/（−）
- 牙周探诊深度
- 附着丧失
- 根分叉病变
- 松动度

无牙周袋底探诊出血

检查的第一个工作就是判断是否存在牙周袋探诊出血。

为什么要进行 BOP 检查？

牙周袋探诊出血仍然被认为是疾病活跃性的最佳的独特的临床指征（图 8-1）。从病史上说，BOP 被认为是该时该位点发生活跃性疾病的指征。实际上它认识到位点特异性这一概念

sites to undergo a detailed examination need to be very carefully observed owing to the site-specific nature of the disease (see Chapter 5). No assumptions can be made about the mouth as a whole from a limited examination of only a few sites.

The detailed clinical examination comprises five separate parts and always precedes any radiological examination or other special tests. The information gained from this allows the clinician to decide on a logical basis whether additional tests are required and if so which ones. This ensures that only necessary tests are prescribed. The five components are:

- presence/absence of BOP
- PPD
- LOA
- furcation involvement
- mobility.

Lack of Bleeding on Probing from the Base of the Pocket

The first thing to be examined is whether there is or is not bleeding on probing from the base of the pockets.

Why?

Bleeding on probing from the base of the pocket is still regarded as the best individual clinical indicator of disease activity (Fig 8-1). Historically, BOP from the base of the pocket was thought to indicate the presence

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图 8-1　牙周袋探诊出血

Fig 8-1　Photograph illustrating bleeding on probing from the base of the pocket.

的同时承认疾病在本质上常常是间断发作的。然而在 20 世纪 90 年代早期，研究证实最多只有 30% 的 BOP 位点继续失去牙周附着。因此更容易接受现在谈到的没有牙周袋探诊出血，因为我们知道几乎 100% 的这样的位点在非吸烟人群不会发生附着丧失。无牙周袋底探诊出血与无疾病活跃性是相关的，但是这项研究没有考虑吸烟因素。故在当前吸烟人群中个别位点无 BOP 的情况不包括在内。

of active disease at that site at that time. Indeed this mantra recognised the concept of site specificity while acknowledging that disease is often episodic in nature. However, in the early 1990s, research demonstrated that only a maximum of 30% of sites exhibiting BOP from the base of pockets went on to lose attachment. It is thus more acceptable now to talk about lack of bleeding on probing from the base of the pocket, since we know that almost 100% of such sites in non-smokers will not progress to attachment loss. Whilst a lack of bleeding from the base of the pocket is associated with a lack of disease activity, the studies that demonstrated this did not take smoking habit into account. The absence of BOP from individual sites in current smokers cannot therefore be included in these interpretations.

怎样进行牙周探诊？

BOP 的评估必须以系统的方式进行以保证所有位点都能被检查到。所

How?

The assessment of BOP must be approached in a systematic manner ensuring

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以每次检查依照同样的规则和同样的方法进行显得尤为重要，例如，检查下牙弓颊面时从左下颌第二磨牙开始沿着牙弓逐颗牙齿进行检查，对应上颌牙弓也按此顺序进行。完成后在舌/腭面重复进行检查。检查最好是将牙周探针沿着适宜的龈沟底进行"爬行"探查并检验是否有出血。

that all sites are examined. It is thus important to get into a routine of performing this test in the same way each time, e.g. checking all buccal surfaces of the lower arch starting at, say, the lower left 7, progressing round the arch and then doing the equivalent for the upper arch. Once completed, this is repeated for the lingual/palatal surfaces. The examination is best accomplished by running a periodontal probe gently along the bases of appropriate gingival sulci and then checking to see whether bleeding occurs.

由于使用牙周探针尖端点的压力会穿通局部软组织导致假阳性结果，所以保证探针尖端的移动是很重要的。尽管所有诱发的出血将出现在龈缘，但本质都是被检查的牙周袋底或龈沟底的组织，有时出血是即刻出现出现的且出血多，有时出血是延迟出现的且出血量少。所以建议完成颊侧或舌侧的检查后，临床医师应该再次观察已检查的位点区域以便检查延迟的探诊出血。探诊出血量与是否有探诊出血相比，其重要性要低得多，所以许多检查者选择从质上记录BOP，如出现或不出现。尽管这样可能在初期降低了详细信息的价值，但却避免了对更复杂指数的解释并节约了时间。

It is important to keep the probe tip moving as applying point pressure may result in false positive results due to localised soft tissue penetration. Although any bleeding induced will be noticed at the gingival margin, it is essential that it is the tissues at the base of the pocket/gingival sulci that are tested. Sometimes the bleeding is immediate and profuse, while at other times it is delayed and scanty. It is suggested, therefore, that once a buccal or lingual quadrant has been completed, the clinician looks back along the quadrant sites tested so that delayed bleeding may be detected. The amount of bleeding produced is much less important than whether or not it occurs at all. It is because of this that many examiners choose to record BOP from sites qualitatively as being either present or absent. Although this may initially appear

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to be undervaluing detailed information, it does remove the problems of individual interpretation of more complex indices and the associated time involved.

信息记录

这可通过各种方法完成，但最简洁的方法可能是用图表如图 8-2 所示。首先所有的缺失牙被删除（在代表牙齿的框内划一条横线来说明）且余留牙被分为 4 个面——颊侧面、舌侧面、近中面和远中面。标记每一位点牙周袋底探诊出血与否，习惯性地用红色来标记相应的部分。这种记录形式易于计算感染位点的百分比也使它们的分布形象化。这些信息对治疗初期和再次评估期的治疗计划都非常有价值，同样对患者的治疗动机也很有帮助。

Recording the Information

This may be done in a variety of ways, but perhaps the most succinct is via the type of chart shown in Fig 8-2. First, all missing teeth are deleted (illustrated by placing a horizontal line through the box representing the tooth) and those present are divided into 4 surfaces - buccal, lingual, mesial and distal. Presence or absence of BOP from the base of the pockets at each site is then noted and the appropiate segments coloured in - conventionally in red. Once completed, this format allows easy calculation of the percentage of sites affected and also allows pictorial representation of their distribution. This information is useful for both treatment planning at the beginning of therapy

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探诊出血点 (SITES WITH BLEEDING ON PROBING)

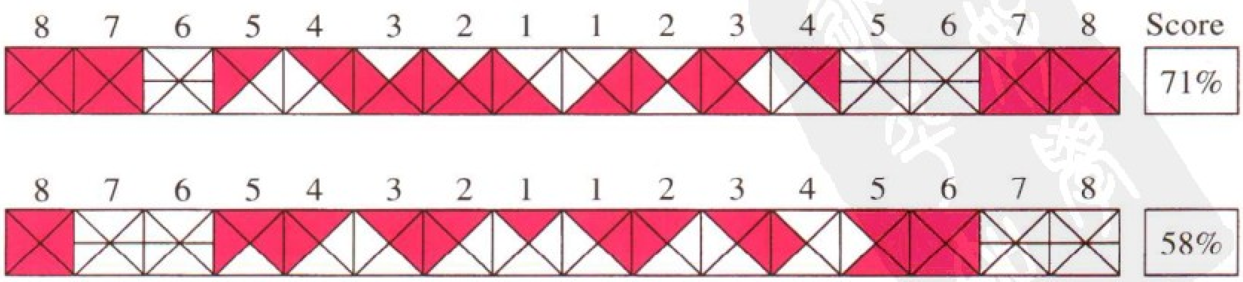


图 8-2　牙周出血图表

Fig 8-2 Photograph showing a completed bleeding chart.

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and at the re-evaluation phase, as well as being useful in patient motivation.

牙周袋探诊深度（PPD）

口内尽可能多的位点的牙周袋探诊深度的测量是最基本的。由于患者间或患者人群内牙周疾病经历的变化，应该强制性地对多个位点进行测量。

Probing Pocket Depth (PPD)

Measurement of PPDs at as many sites as possible around the mouth is essential. Multiple site measurement is mandatory owing to the variation in periodontal disease experience seen within and between mouths.

为什么要进行 PPD 检查?

如果牙周袋≤3 mm，患者基本上能通过正常的家庭护理方法保持位点的清洁。如果牙周袋超过这一深度，患者就不能有效地清洁，进而增加继续破坏的危险。所有牙周治疗的一个简单的目的可被认为是使所有 PPDs ≤ 3 mm。

Why?

In basic terms, if a pocket is ≤3mm, patients can keep the site clean using normal home-care methods. If pockets are deeper than this, patients cannot adequately clean them, resulting in an increased risk of continued destruction. One simple aim of periodontal therapy may therefore be regarded as reducing all PPDs to ≤ 3mm.

怎样探诊牙周袋深度?

当进行临床探诊评估时，PPD是从龈缘到牙周袋底的距离（第 1 章）。测量是使用许多不同牙周探针（图 8-3）中的任意一种，这些探针的共同性特征是有一个降低穿透组织危险的钝头和探针上的一系列方便测量的标记。

How?

PPD is the measurement from the gingival margin to the base of the pocket, when assessed by clinical probing (Chapter 1). It is measured using any one of a number of different periodontal probes (Fig 8-3) which all share the characteristics of having a blunt end to reduce the risk of tissue penetration and a series of markings along their length to facilitate the measurement process.

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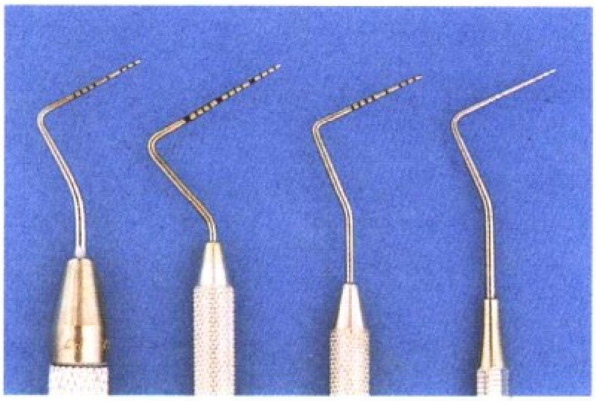


图 8-3　几种牙周探针

Fig 8-3 Photograph illustrating several types of periodontal probe.

信息记录

一些有经验的医师发现有序地记录左上象限颊侧的PPD，再记录左上象限腭侧的 PPDs 等，用红笔圈出探诊出血位点，用蓝/黑笔圈出溢脓的位点是比较快的方法。这也清楚地提供了每牙六个位点BOP评估，但必须假定使用正确的探诊压力。不管用何种系统均依赖于个人临床喜爱。如果临床医师希望得到详细记录，如图 8-4 所示的"双重牙周图表"很有帮助。如何完成这种牙周袋图表的特殊细节在本章后面详述。

Recording the information

Some experienced clinicians find it quicker to record PPDs sequentially as buccal maxillary left quadrant, followed by palatal maxillary left quadrant, etc., and to circle sites that bleed on probing in red pen and sites of suppuration in blue/black pen. This clearly also provides six-sites-per-tooth assessment of BOP, but assumes the correct probing pressure has been used for bleeding scores. Whichever system is used depends on personal clinical preference. Should the clinician wish to have detailed records, a "double periodontal chart" such as that shown in Fig 8-4 is helpful. Specific details of how to complete this type of pocket chart are given later in this chapter.

附着丧失

测量尽可能多位点的附着丧失是很重要的。

LOA

Measurement of LOA at as many sites as possible around the mouth is important.

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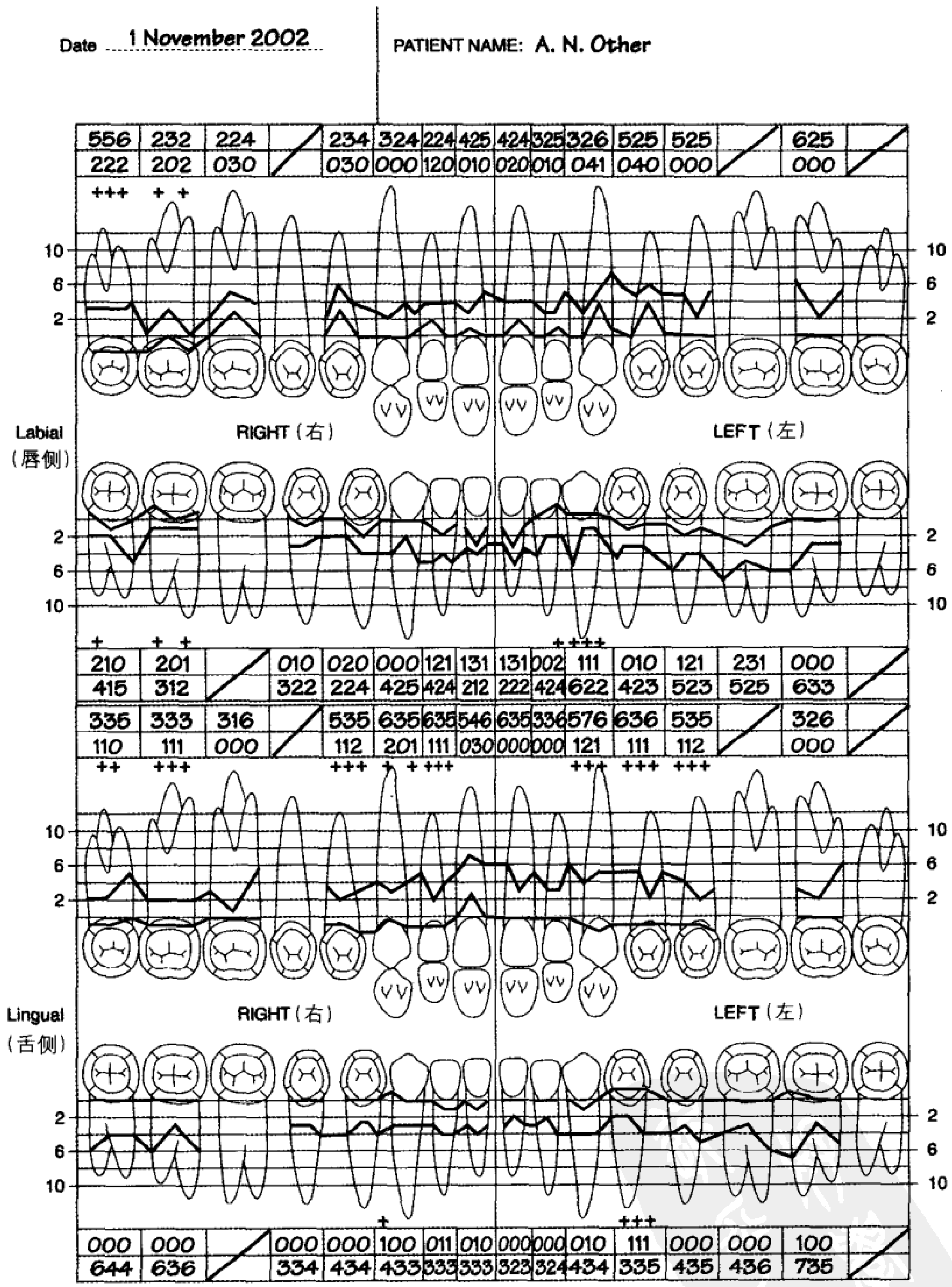


图 8-4　完成的“双重牙周检查图表”

Fig 8-4 Completed “double periodontal chart”.

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为什么要测量 LOA？

LOA 是牙萌出后特定位点全部牙周破坏的量度。但是它没有提供已发生疾病的发生次数和出现时间的信息。在检查、诊断和治疗计划上，它应该被认为是背景，当前牙周袋深度的详细情况应以此为背景。

Why?

LOA is a measure of the sum total of periodontal destruction experienced at a particular site since the tooth erupted. It does not, however, provide information about the number of episodes of disease that have taken place or when they occurred. In terms of examination, diagnosis and treatment planning, it should be regarded as the background upon which the current detail of pocket depth should be placed.

怎样测定 LOA？

LOA 是测量釉牙骨质界（ACJ）到牙周袋底的距离。

How?

LOA is the measurement from the amelocemental junction (ACJ) to the base of the pocket.

测量 LOA 的重要性？

牙周袋深度和 LOA 常常是不同的——只有当龈缘位于 ACJ 时它们才相同，牙龈肥大位点（图 8-5）的牙周袋深度大于 LOA，此时仅依靠牙周袋深度作为过去疾病经历的指征将过高的评估疾病的严重性。相反，当牙龈退缩时（图8-6），仅依靠牙周袋深度观察者将低估疾病的经历。

Significance?

Usually pocket depth and LOA are different—only if the gingival margin lies at the ACJ will they be the same. In sites with gingival enlargement (Fig 8-5) pocket depth will exceed LOA and reliance upon pocket depth alone as an indicator of past disease experience will lead to over-estimation. The opposite is true in sites exhibiting recession/shrinkage (Fig 8-6), where using pocket depth alone would lead the observer to under-estimate disease experience.

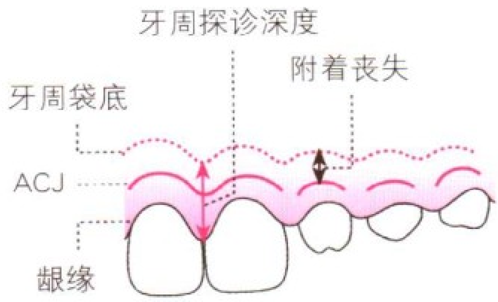
信息记录

下面讨论的"双重牙周图表"是鉴

Recording the information

The "double periodontal chart" discussed

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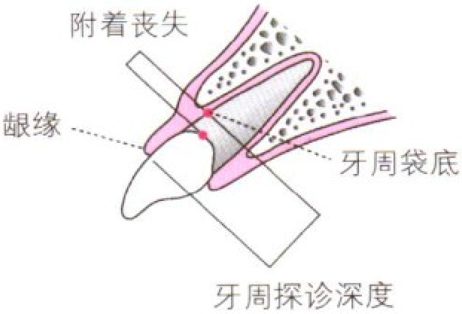




图 8-5　牙龈肿大病例以及 LOA 和 PPD 测定方法（本例 PPD > LOA）

Fig 8-5 Photograph of gingival enlargement and diagram to show how both LOA and PPD would be measured. In this case, the PPD is greater than the LOA

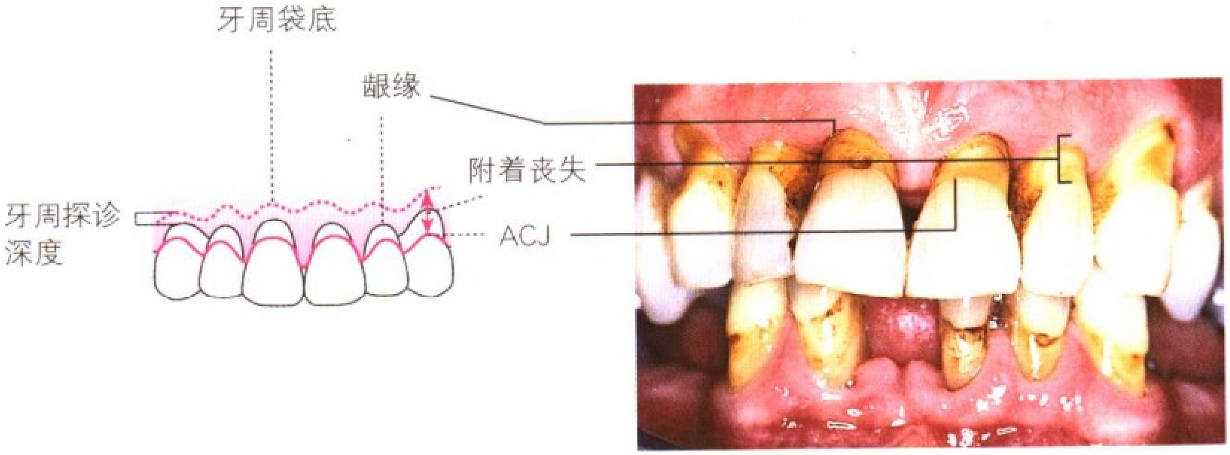


图 8-6　牙龈退缩病例以及 LOA 和 PPD 测定方法（本例 PPD < LOA）

Fig 8-6 Photograph of gingival recession and diagram showing how both LOA and PPD would be measured. In this case, the PPD is less than the LOA

定这些信息的一个好方法。

below is a good means of identifying this information.

根分叉病变

指多根牙的牙根分叉部位牙周支持组织的水平丧失。

Furcation Involvement

This refers to the recognition of horizontal loss of support in the areas where the roots of multi-rooted teeth converge.

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重要性?

因根分叉病变可降低治疗的预后，所以确定是否有根分叉病变非常重要，其原因有二：

1. 医师和患者都难于进入病变位点从而进行清洁，所以这些位点的疾病的控制和复发的预防都比较困难。

2. 受累牙可能出现牙髓活力丧失。这是由于从髓腔到根分叉区的副根管成为细菌及其产物进入髓腔的管道，这种对牙髓的攻击方式常常是没有症状的。

Significance?

It is important to check for furcation involvement as this may reduce the prognosis for a tooth for two reasons:

1. The involved sites are difficult for both the clinician and the patient to gain access to and thus to clean. Thus controlling disease and preventing recurrence at such sites is less likely.
2. Loss of vitality of the involved tooth/teeth may occur. This happens because accessory canals running from the pulp chamber to the furcation area act as a conduit for bacteria and their products. Assault on the pulp in this manner is often symptom free.

探查?

是最基本的临床检查方法，使用一种弧形根分叉探针能方便地检测牙周支持组织的水平丧失。需要检查的牙齿有：

- 上颌第一前磨牙常有一个颊根和一个腭根，所以根分叉病变的探查应该从近中和远中面进行。

- 下颌磨牙常有一个近中和一个远中根，所以根分叉病变的探查应从颊侧和舌侧进行。

Detection?

Clinical examination is essential and is facilitated using a curved furcation probe to measure horizontal loss of support. The teeth to check are as follows:

- *Maxillary first premolars* usually have a buccal and a palatal root so clinical detection of furcation involvement should be from both the mesial and distal aspects.
- *Mandibular molars* usually have one mesial and one distal root so clinical detection of furcation involvement should be from both the buccal and lingual aspects.

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• 上颌磨牙常有一个大的腭根和一个近中颊根以及一个远中颊根。临床检查这些牙齿的根分叉病变时，探查应从两颊侧牙根之间的颊侧部分，从近中部分（从外展隙腭侧进入）和远中部分（可从外展隙任一侧进入）。

• *Maxillary molars* usually have one large palatal root and both a mesio-buccal and a disto-buccal root. Clinical examination to detect the presence of furcation involvement in these teeth should be from the buccal aspect between the two buccal roots, from the mesial aspect (approaching from the palatal side of the embrasure) and from the distal aspect (which may be approached from either side of the embrasure).

根分叉病变严重程度分级

这对于根分叉病变的最终治疗非常重要。有许多分级系统，Hamp 等（1975）提出的分级系统，因其简单而实用而被推荐使用，该系统如下（图8-7）：

1 度 = 牙周支持组织水平丧失不超过牙齿颊舌径的1/3。

Grading the severity of furcation involvement

This is important as the definitive treatment of a furcation lesion depends upon the degree of involvement. There are many classification systems but that suggested by Hamp *et al*. (1975) is to be commended for its simplicity and pragmatism. In this system (Fig 8-7):

Degree 1 = horizontal loss of periodontal support not exceeding one-third of the bucco-lingual width of the tooth.

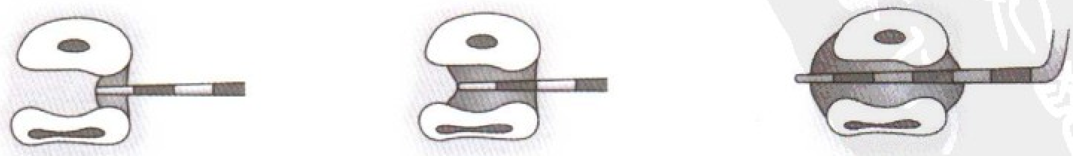


图 8-7 根分叉病变分度

Fig 8-7 Diagram illustrating the different degrees of furcation involvement.

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2度＝牙周支持组织水平丧失超过牙齿颊舌径的1/3，但尚未与对侧贯通。

3度＝根分叉区牙周组织水平"彻底贯穿"破坏。

Degree 2 = horizontal loss of periodontal support exceeding one-third of the width of the tooth, but not encompassing the total width of the furcation area.

Degree 3 = horizontal "through and through" destruction of the periodontal tissues in the furcation area.

根分叉病变报告

临床医师应该经常注意哪个牙齿出现根分叉病变，哪个/些根分叉受影响以及每个感染位点的病损程度。

Reporting furcation involvement

The clinician should always note which tooth has furcation involvement, which furcation(s) is/are affected and the degree of involvement present at each affected site.

鉴别诊断

除了牙周病引起根分叉病变外，根分叉病变可能与始发于根管的疾病有关，或可能是𬌗负荷过大的结果。所以治疗不应该在最终诊断作出前开始。

Differential diagnosis

In addition to being caused by periodontal disease, a lesion in the furcation area may be associated with problems originating from the root canal or be the result of occlusal overload. Treatment should not therefore be initiated until a definitive diagnosis has been made for the lesion.

活力测试

对所有涉及根分叉病变的牙齿建议进行活力测试。因为这些牙齿可能出现活力丧失——经常是无症状的。副根管感染可能发生这种情况（见图8-8）。根分叉区菌斑中的毒素通过这些根管的距离很短，并最终使一个或更多根管内的牙髓组织失去活力。自然，如果出现这种情况，将不可避免地要对感染牙进行根管治疗或拔除感染牙。

Vitality testing

It is advisable to "vitality-test" all teeth displaying furcation involvement. This is necessary because loss of vitality can occur in such teeth – often without symptoms. This may happen owing to infection of accessory canals (see Fig 8-8). Toxins from plaque lying in the furcation area have a very short distance to travel through these canals to reach, and ultimately render non-

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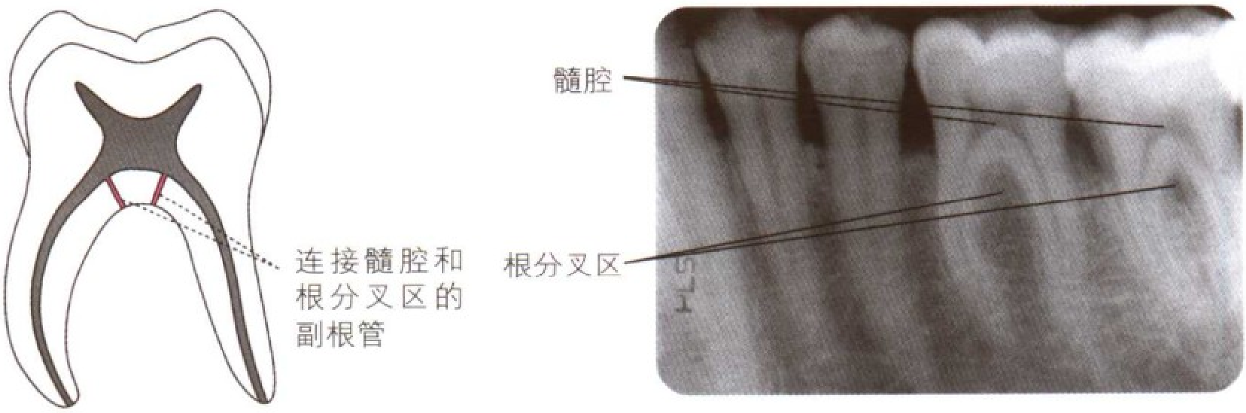


图 8-8 X线片示髓腔与根分叉区接近，线图示连接两者的副根管

Fig 8-8 Radiograph illustrating the proximity of the pulp chamber to the furcation area, and a diagram showing stylised accessory canals linking the two.

牙齿松动度

所有牙齿均应该检查松动度，其在监控每次就诊时松动程度的变化以及制定治疗计划都很重要。很明显，如果一个牙表现为2度松动，在经过数年后仍然如此，相比较一个牙在3个月前没有松动度而现在出现2度松动，我们更应多关心后者。总的来说松动度越大，预后越差。

怎样检查松动度?

使用两个手用器械柄，选择从颊侧或舌侧，也可沿牙齿长轴垂直向加力于牙冠上来检测松动度。

vital, pulpal tissue in one or more canals. Naturally, if this occurs, either root canal therapy or extraction of the affected tooth will be necessary.

Tooth Mobility

Tooth mobility should be checked for all teeth and is important for monitoring changes in the degree of mobility from visit to visit and also to inform treatment planning. Clearly, if a tooth displays degree 2 mobility and has done so for a period of years, this is of less concern than one which now has this degree of mobility but 3 months earlier exhibited no mobility. In general, the higher the mobility the poorer the prognosis.

How?

The handles of two hand instruments are used, and pressure is placed on the crown of the tooth to be tested, alternately from

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牙齿松动分级

很遗憾，分级是很主观的，但是使用一个分级系统总比使用非常不具体的术语如"轻微"或"中度"要好，因为它们对不同人具有不同的含义。有很多分级选择，但下面介绍一种简单实用的分级系统：

0度=无可探查到的动度/生理动度（传统认为最高0.2 mm）。

1度=牙冠水平向动度为0.2～1 mm。

2度=牙冠水平向动度>1 mm。

3度=牙冠水平向和垂直向均有动度。

牙齿松动的原因

除了检查牙齿松动程度外，应该考虑其原因，因为有效的治疗依靠对致病原因的准确诊断，牙齿松动有三种原因，它们可单独作用或联合作用：

- 创伤——直接钝性创伤或是𬌗创伤
- 根尖疾病
- 导致牙周支持组织丧失的牙周疾病

the buccal and lingual aspects and also vertically down the long axis of the tooth.

Grading of tooth mobility

Unfortunately, grading is rather subjective, but using a system is better than using very imprecise terms such as "slightly" or "moderately", as these have different meanings to different people. There are a number of choices but a simple and practical one is documented below:

Degree 0 = no detectable movement/ physiological mobility (classically up to 0.2mm).

Degree 1 = mobility of the crown of the tooth 0.2-lmm in the horizontal direction.

Degree 2 =mobility of the crown of the tooth >lmm in the horizontal direction.

Degree 3 = mobility of the crown in both the horizontal and vertical planes.

Causes of tooth mobility

This should always be considered in addition to examining the degree of involvement, because effective treatment depends upon an accurate diagnosis of the cause(s). There are three main causes, which may apply singly or in any com-bination:

- trauma – either direct blunt trauma or occlusal trauma
- periapical disease
- periodontal disease leading to loss of support.

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信息记录

最佳方式是通过图表进行信息记录如图8-9所示。每个框代表一颗特定牙齿，删除缺失牙，按上面牙齿松动度从0到3分级，将其填入对应的框内。

Recording the information

This is best done using a chart such as that in Fig 8-9. Each box represents a particular tooth and after deletion of missing teeth the mobility score, graded from 0 to 3, as above, is placed within the appropriate box.

"双重牙周图表" 记录

为什么?

初始图表记录了基线牙周状况，后面的图表则记录经过一段时间治疗后特定位点测量值的变化。

Recording a "Double Periodontal Chart"

Why?

Initial charts provide a record of the baseline periodontal status and subsequent charts demonstrate how measurements change at specific sites over time with treatment.

怎样进行记录?

双重牙周图表的完成需要有椅旁助手。

How?

This exercise requires the presence of a chairside assistant to record the data.

松动度　　**姓　名：** A. N. Other　　**日　期：** 1 November 2002

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
1	2	—	2	1	○	○	1	1	○	○	1	—	—	1	1

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
2	—	—	1	○	○	○	○	1	○	○	○	1	1	—	—

图 8-9　完成的牙齿松动度图表

Fig 8-9　Completed mobility chart.

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程序包括：

1. 记录缺失牙并从图表中删除。

2. 记录龈缘测量值（常用红笔记录），确信助手知道你开始测量的位置，如左上颌第二磨牙颊侧，然后用合适的有条带刻度探针以毫米（mm）为单位测量龈缘到ACJ的距离，ACJ可用目视或触觉进行探查。如果龈缘位于ACJ的冠方则从探针上读取的"mm"读数记录为正值（如+2；图8-10），当龈缘位于ACJ根方时可简单地记录其读数（既不用正也不用负）（图8-11）。

The procedure is as follows:

1. Record all missing teeth and delete them from the chart.
2. Record the gingival margin measurement (usually recorded in red). Ensure that the assistant knows exactly where you are starting from, e.g. buccal upper left 7, and then measure the distance in millimetres (mm) from the gingival margin to the ACJ using a suitable banded probe. The ACJ may be detected either visually or by touch. The "mm" reading from the probe is recorded as a plus if the gingival margin lies coronal to the ACJ (e.g. +2:

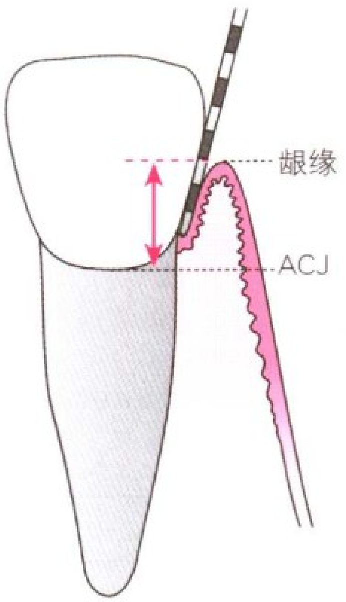


图8-10 牙龈肿大时龈缘到ACJ距离的毫米测量值为"+"

Fig 8-10 Diagram illustrating a periodontal probe being used to measure the distance of the gingival margin to the ACJ in a site of gingival enlargement. The measurement in mm is recorded as a "plus" score.

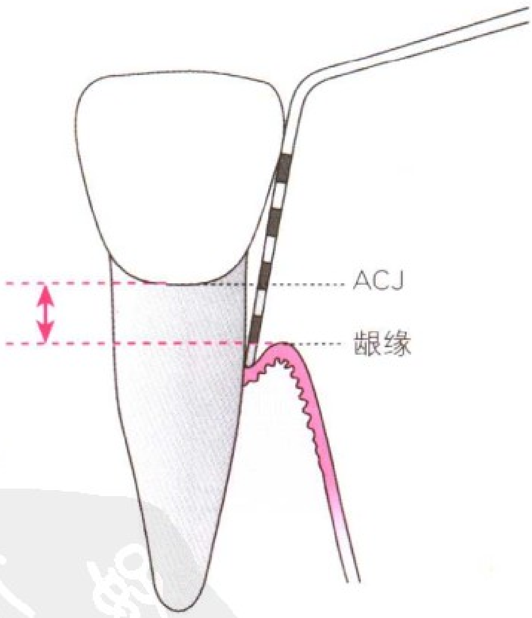


图8-11 牙龈退缩位点的牙周探测，测量值不加"+"或"–"

Fig 8-11 Diagram as above except in a site exhibiting gingival recession. The measurement is given in mm only with no "plus" or "minus" designation.

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Fig 8-10), while it is recorded as simply the number of mm (with neither a plus nor a minus) when the gingival margin lies apical to the ACJ (Fig 8-11).

3. 每牙颊面记录三个测量值（如远中颊、颊和近中颊），并填入图表中相对应的框内（图 8 - 12）。

3. Three measurements are recorded for the buccal surface of each tooth (i.e. disto-buccal, mid-buccal and mesio-buccal) and the appropriate results are placed in the corresponding boxes on the chart (Fig 8-12).

4. 记录采用系统方式以保证没有牙或位点缺失。但是由于可能出现的无意识的错误，建议记录到中线位置时说明一下，当牙医与助手之间存在不一致时，这会让双方明白并得到及时纠正。

4. The recording occurs in a systematic manner to ensure that no teeth or sites are missed. However, it is possible that mistakes can unwittingly occur and it is advisable on reaching the midline to state this. Should there be a discrepancy between the dentist and assistant, then it will become clear at this stage and may be rectified immediately.

5. 检查记录完上下牙弓颊面后，再继续以同样的系统方式记录腭／舌面。

5. Once the buccal surfaces of both arches have been examined, recording of data from the palatal/lingual surfaces may proceed. This is completed in the same systematic manner.

6. PPD 测量值的记录常用蓝笔。PPD 的值是使用探针测量龈缘到牙周袋底的距离。如上所述，该测量值包括围绕每颗牙的 6 个位点——颊侧3个，舌／腭侧 3 个。

6. The PPD measurements are then recorded (usually in blue). This is done using the probe to measure the distance from the gingival margin to the base of the pocket. As described above, this data is retrieved from a total of six sites around each tooth – three buccal and three lingual/palatal.

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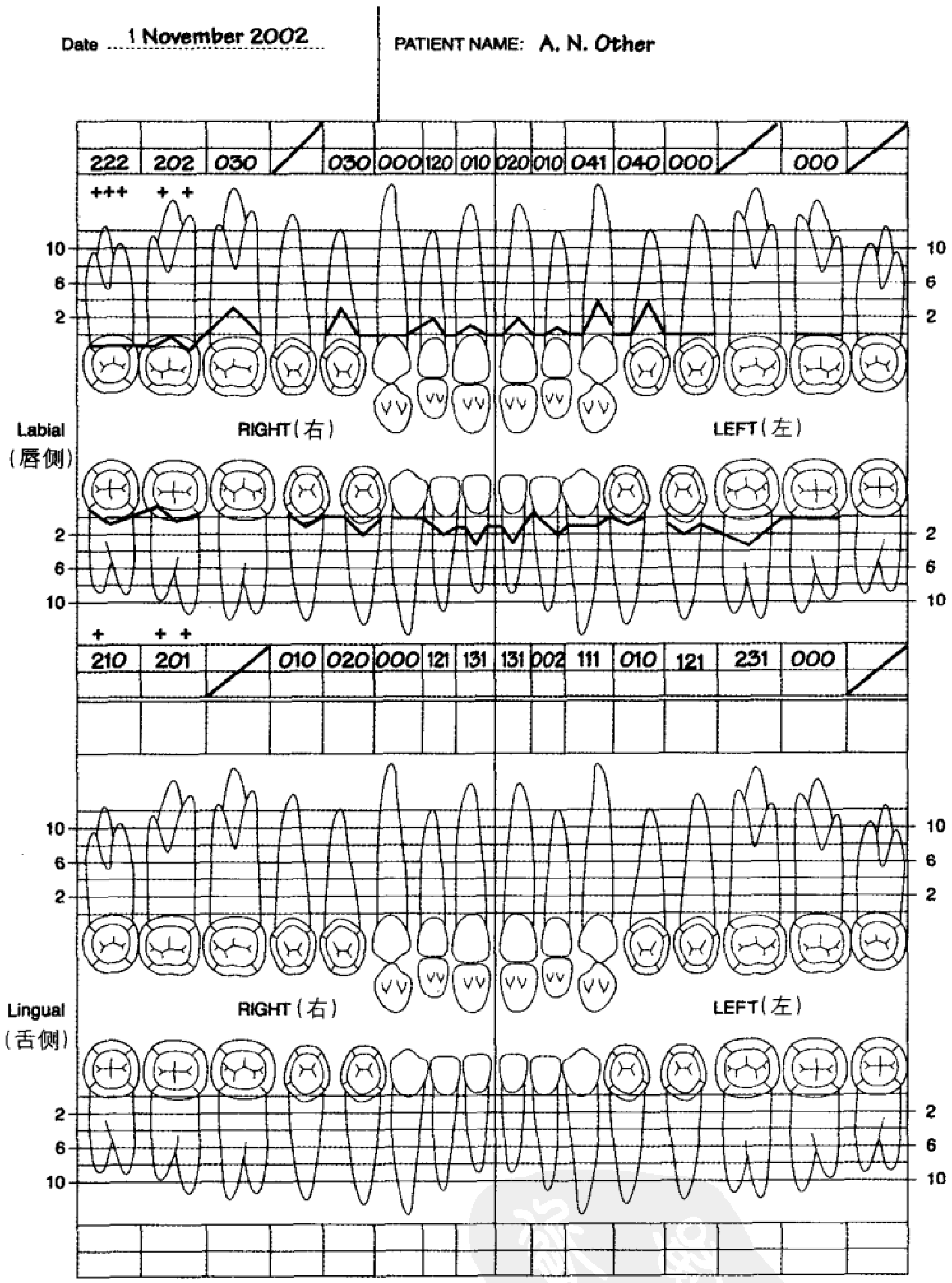


图 8-12　部分完成的“双重牙周检查图表”尖牙用“/”显示，测量唇颊面 ACJ 到龈缘的毫米值并标记在上图正确位置，红色连线代表龈缘位置

Fig 8-12 Partially completed "double periodontal chart" showing missing teeth ⍁ and the measurements in mm from the ACJ to the gingival margin on the buccal/labial surfaces of the teeth. The measurements are then transferred to lie on top of the drawings of the teeth in the correct position and the red line representing the position of the gingival margin is generated by joining them up.

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7. 当所有的数据填入图表框内时，"全口图"变得清晰。有的临床医师喜欢结合图表中的所有特征得出一个粗略的视觉总结。这时，首先常常完成龈缘线，通常用红笔描绘。根据记录的牙周袋深度距离代表龈缘线的红线画一条蓝色线代表牙周袋底（图8-4）。但重要的是应该认识到这条线只代表记录要点之间附着轮廓的估计，并不是真的准确情况。

7. Once all the appropriate figures have been filled in on the chart boxes the "whole mouth picture" becomes clear. Some clinicians prefer to join all the points drawn on the chart, to provide a rough visual summary. In this situation, the gingival margin line is usually completed first and by convention is depicted in red. The line representing the base of the pocket is then drawn in (by convention, in blue) by measuring the distance recorded for pocket depth from the red line representing the gingival margin (Fig 8-4). However, it is important to recognise that such lines represent an estimate of the attachment contour between recorded points and are not truly accurate.

优　点

完成的图表可得到全口每颗牙 6 个点的下列检查。

1. PPD是龈缘到牙周袋底的距离。
2. LOA是ACJ到牙周袋底的距离。
3. PPD和LOA贯穿整个口腔，后者由于揭示了附着丧失的严重程度和分布，对于确定诊断有很大的帮助。
4. 牙龈肿胀／退缩的发生和位置

Advantages

The completed chart allows examination of the following at six points around each tooth in the whole mouth.

1. PPD measured from the gingival margin to the base of the pocket.
2. LOA measured from the ACJ to the base of the pocket.
3. The pattern of both PPD and LOA throughout the mouth. The latter is very helpful in reaching a periodontal diagnosis as both severity and distribution are exhibited.
4. The presence and location of gingival

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得到证实（从ACJ到龈缘的距离）。

enlargement/recession is demonstrated (the distance from the ACJ to the gingival margin).

缺　点

除了用一条线连结测定点得出的假设外，双重牙周图表有两个主要缺点：

1. 由于所有测量是基于探针探查所得，它可导致过低或过高估计测量值（这里将不再讨论），所以存在固有的不精确的危险。
2. 这一过程需要大量时间，尽管经验能减少用时。任何情况必须权衡它与所获信息之间的利弊。

在实际工作中计算机软件程序简化了牙周图表，但很多是有限的，不能得到附着丧失的记录。

Disadvantages

There are two main disadvantages of double periodontal charts in addition to the assumptions made when the measured points are linked using a line:

1. There is an inherent risk of inaccuracy with all measurements based on probing, which can lead to under- or overestimation of measurements. This is discussed below.
2. The procedure takes considerable time, though this reduces with experience. In any case this has to be weighed against the information gained.

Many computerised software programs facilitate periodontal charting in practice, but many are limited and do not allow the recording of attachment loss.

与手工探查有关的误差

产生的测量值明显是操作者感受的，几个因素影响结果的准确性，它们包括：

1. *探诊的力量*。理想的探诊力量应该为0.2～0.5N（标准探针用20～25g力），尽管我们知道使用的探诊力量变化很大，甚

Errors Associated with Manual Probing

The measurements produced are clearly operator sensitive and several factors affect the accuracy of the results gained. These include:

1. *The probing force applied.* Ideally this should be around 0.2-0.25N (20-25g weight for typical probes), though it is known that large variations arise in

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至同一检查者在同一口腔内不同位点使用的探诊力量均有较大变化。过大的探诊力量会穿透牙周袋底的组织并过高估计牙周袋探诊深度。而过小的探诊力可能意味没有接触袋底而过低估计牙周袋深度。压力敏感探针不允许大于设定的最大值（图8-13），但是在低于最大值时，它并不能预防探诊力量的变化。

*2．探针的大小和形状。*厚或宽的探针不能通过窄或弯曲的牙周袋并导致低估探诊深度。同样，尖锐的探针，即使使用适宜的

the probing force applied even by the same operator at different sites in the same mouth. Excessive force will result in penetration of the tissues at the base of the pocket and thus overestimation of the true probing pocket depth occurs. Alternatively, application of too little force may mean that the base of the pocket is not reached and pocket depth underestimation may result. Pressure-sensitive probes have been developed which do not permit the application of forces greater than a set maximum (Fig 8-13). They do not, however, prevent variation in probing forces which may be applied below that maximum.

2. *The size and shape of the probe.* A thick or broad probe will not be able to negotiate a narrow or tortuous pocket and may result in underestima-

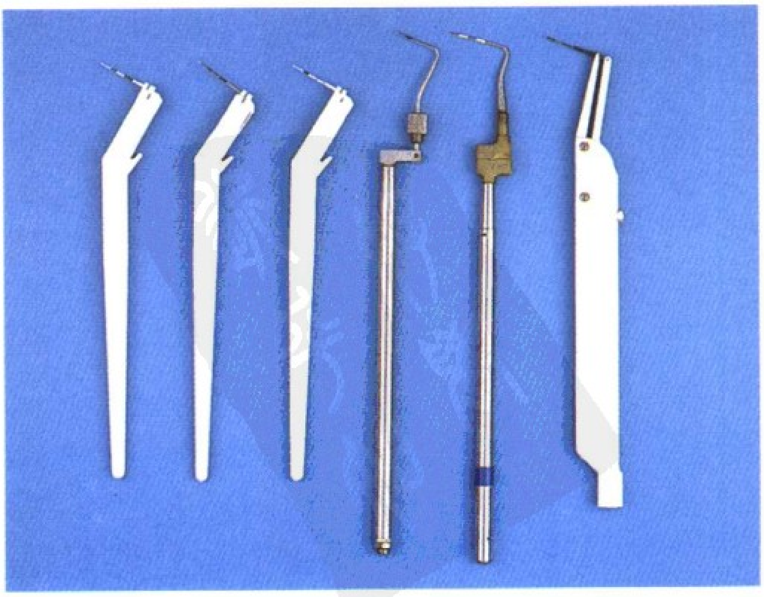


图 8-13　压力敏感探针

Fig 8-13 Photograph of a selection of pressure-sensitive probes.

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压力，也能轻易穿透袋底软组织而高估牙周袋深度。

3. *组织的炎症状态*。如果袋底是感染的，袋底结缔组织中高百分比的胶原丢失，组织的抵抗力较弱，即使正常的探诊力量也常导致组织穿通而高估牙周袋深度测量值。

4. *牙石、外形过大的修复体、球状牙冠和不利的牙根形态*。患者初次治疗时普遍存在牙石，这些沉积物可阻碍探针到达牙周袋底。这可导致低估牙周袋深度而使初次检查时出现误差，并形成治疗后牙周袋明显加深的印象，去除这些沉积物后则易于接近牙周袋底。外形过大的修复体，鳞茎状牙冠和非常弯曲的牙根同样能阻碍探针通过牙周袋而导致相同的潜在问题。

tion of probing depth. Equally, a sharp probe, even applied at appropriate pressure, could easily penetrate the soft tissues at the base of the pocket and result in overestimation.

3. *The inflammatory state of the tissues.* If the base of the pocket is inflamed, a high percentage of the collagen in the connective tissue at the pocket base is lost. This means that the tissue has poor resistance and application of a probe, even at normal forces, will often result in tissue penetration and thus overestimation of pocket depth measurements.

4. *The presence of calculus, over-contoured restorations, bulbous crowns and unfavourable root morphology.* Calculus is very common when patients first present for treatment and these deposits may prevent negotiation of the probe to the base of the pocket. This results in underestimation of pocket depths introducing errors at the time of the initial examination, and also giving the impression of apparent deepening of pockets after therapy, when these deposits have been removed facilitating access to the base of the pocket. Presence of over-contoured restorations, bulbous crowns and very curved roots also prevent adequate negotiation of the pocket by

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the probe, with similar resultant potential problems.

5. *探针的角度*。如果探针放置时没有与牙长轴平行可能会高估牙周袋深度。

5. *The angle of the probe*. If the probe is placed at an angle other than parallel to the long axis of the tooth this may result in overestimation of the pocket depth.

6. *探诊位置变化*。连续测量时，如果探针每次不是准确地放在同一点，则测量值很难相互比较。临床试验中，可使用殆垫来降低这种误差（图 8-14）。

6. *Variation in probing position*. If sequential measurements are made and the probe is not introduced at precisely the same point each time, comparative measurements are very difficult. In clinical trials, occlusal stents are used to reduce this source of error (see Fig 8-14).

7. *探针上条带的清晰度*。如果探针上的条带不清楚或难以辨认，可能出现操作者误差。

7. *Clarity of banding on the probe*. Making measurements is difficult if the bands on the probe are indistinct and difficult to interpret, which may result in operator errors.

8. *患者耐受性*。耐受性变化较大，如果耐受性低，要接触牙周袋底进行测量比较困难。如果出现这种情况，则可在开始治疗该部位前，通过局部麻醉后分

8. *Patient tolerance*. This varies, and if it is low, reaching the base of the pocket to make measurements can be difficult if not impossible. If this occurs, charting may have to be done

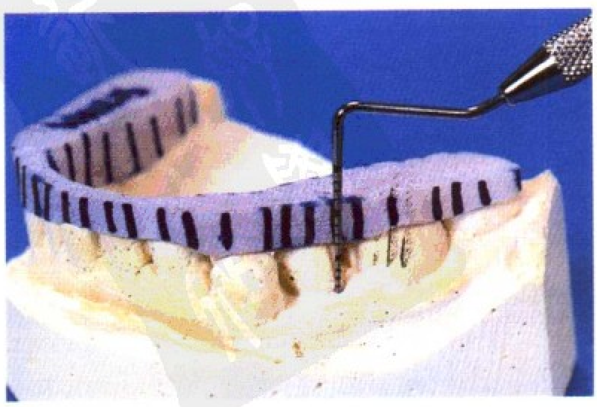


图 8-14 殆垫

Fig 8-14 Photograph of a sten.

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区/象限完成图表。

by sextant/quadrant after administration of local anaesthetic, prior to commencing treatment of that area.

探针选择

现在有手用探针和电子探针两种（图8-15）。电子探针的确能够达到更高的精确度（最高达0.1mm），但电子探针的可重复性并不比手用探针强（图8-16）。总的来说，电子探针有两个主要优点。

- 它记录客观数据，不依靠操作者的视觉。
- 它有一个恒定的探诊力量——常标准化为0.25N。

电子探针昂贵，但现在也有较便宜的使用恒定压力具有标准探针头直径（0.5mm）和清晰条带刻度的手用

Choice of probe

Electronic probes (Fig 8-15) as well as manual probes are now available. The former are certainly capable of greater accuracy of measurement (up to 0.1 mm), but the reproducibility of electronic probing is no greater than that with manual probing (Fig 8-16). In general, electronic probes have two main advantages:

- they permit objective data recording, which is not dependent upon operator eyesight
- there is a constant probing force–usually standardised at 0.25N.

Electronic probes are expensive, however, and manual probes which use a constant force (Fig 8-17) and a standard tip

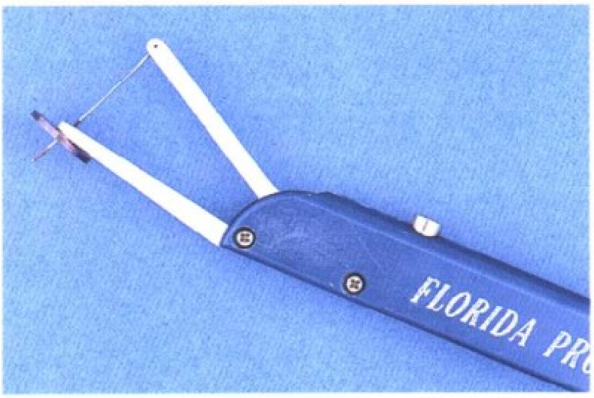


图8-15　Florida电子探针恒定的探诊力量和脚踏自动记录

Fig 8-15 Florida electronic probe, with constant force and automated recording by foot pedal.

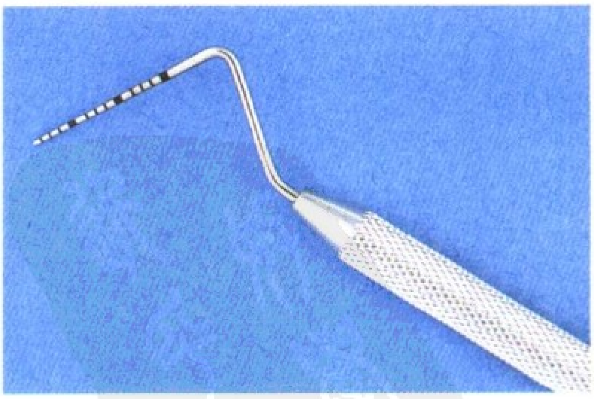


图8-16　UNC-15探针

Fig 8-16 University of North Carolina (UNC-15) probe.

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探诊（图 8-17）。另外，有的临床医师认为塑料探针（图 8-18）更容易探测弯曲牙周袋和不利的外形。必须补充说明塑料探针不利的一面：

- 较差的触觉反馈
- 较易变形
- 由于是一次性使用而需经常性开支，因而是一种昂贵的选择

选择何种探针完全取决于你自己？

关于菌斑指数？

菌斑是牙周袋感染的始动因素，但菌斑指数本身不提供牙周疾病经历的信息。如第 4 章中讨论过的一样，对牙周疾病低易感染性的患者可以表现高菌斑指数，同时，显示很少破坏性疾病的证据，相反情况也可出现。这并不

diameter (0.5mm) with clearer banding are now available at considerably less cost. In addition, some clinicians believe that plastic probes (Fig 8-18) more readily follow tortuous pockets and unfavourable contours. On the downside, it must be added that plastic probes:

- provide less tactile feedback
- deform more easily
- can be an expensive option as the cost is recurrent because they are disposable.

Which probe you choose is entirely up to you!

What about Plaque Scores?

Plaque is the initiator of periodontal inflammation, but by itself the plaque score gives no information about periodontal disease experience. As discussed in Chapter 4, patients with low susceptibility to periodontal disease may display high plaque

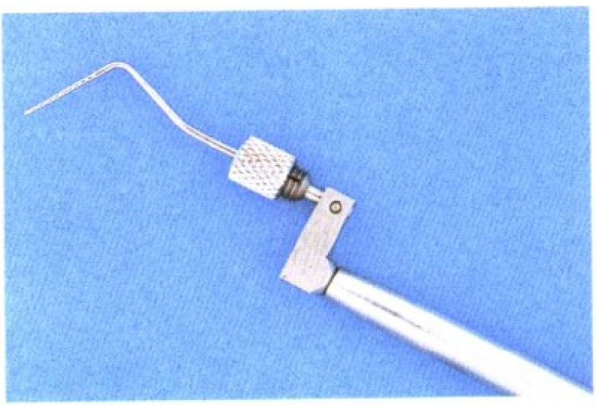


图 8-17　Brodontic 手用持续压力探针

Fig 8-17 Brodontic manual constant force probe.



图 8-18　TPS 塑料持续压力探针

Fig 8-18 TPS plastic constant force probe.

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是说菌斑指数是不相关的——实际上它们在制定治疗计划时起着重要作用，特别是在高易感性个体。但是孤立地说，菌斑指数并没有告诉我们关于口腔内这种疾病的任何信息，而重要的是我们应该认识到大部分患者在牙科就诊前会马上清洁牙齿。

scores, while exhibiting little evidence of destructive disease. The opposite can occur also. This does not mean that plaque scores are irrelevant - indeed they play an important part in treatment planning, particularly for highly susceptible individuals. In isolation, however, they tell us nothing about the disease to be found in a mouth, and it is important to recognise that a majority of patients will clean their teeth immediately prior to a dental appointment.

拓展阅读

Further Reading

1 Galgutt PN, Dowsett SA, Kowolik MJ (Eds.) Periodontics: Current Concepts and Treatment Strategies. London: Martin Dunitz, 2001: Chapter 8

2 Hamp SE, Nyman S, Lindhe J. Periodontal treatment of multirooted teeth (results after five years) J Clin Periodontol, 1975;7:126

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第9章 放射学检查和特殊检验

Radiographic Examination and Special Tests

目 的

本章概括了详细牙周评估过程中使用特殊检查的适应证和禁忌证，论述不同类型的放射学检查和其他特殊检验的优缺点。

Aim

This chapter aims to outline the indications and contraindications for using special tests during a detailed periodontal assessment. Advantages and disadvantages of different types of radiographic examinations are discussed alongside other special tests.

要 点

通过阅读本章，临床医师应该清楚哪种情况下采用哪种X线片，以及如何在牙周评估和放射学报告方面进行解释。

临床检查先于任何其他检测报告并决定是否需要以及需要何种特殊检测。这些特殊检测不是经常要求的。

Outcome

The outcome of reading this chapter will be that the practitioner should be clear about which radiographs to take in which situations, and how to interpret results in terms of a periodontal assessment and document radiographic reports.

The clinical examination precedes any other "hands-on" investigation and determines whether, and if so which, special tests need to be performed. They are not always required.

放射学检查

X线片常用以提供大多数是关于

The Radiographic Examination

Radiographs are used to provide infor-

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硬组织的信息，只在这些数据对已掌握情况提供阳性附加价值时才推荐使用，需要考虑许多要点：

1. 需要打破个体患者在通过X线片中获得额外诊断价值的优点与患者接受辐射的潜在损害之间的平衡。
2. 如果要摄X线片，临床医师必须选择能提供最高诊断价值，产生最少可能辐射的胶片拍摄。
3. 在拍摄的所有X线片的记录中应有一个签名并注有日期的放射报告。
4. 如果X线片很难解释，应该征求放射学家的意见和征求一份书面报告。

技术选择

应该使用快（E-或F-速）胶片和窄的平行X线来尽可能地降低辐射剂量，常规牙科X线片使用三种胶片：口腔全景X线断层片，䍽翼片和根尖片。

mation largely about hard tissues and are recommended only when such data provides positive added value to that already held. There are a number of points to consider:

1. A balance needs to be struck for individual patients in individual circumstances between the advantages of the additional diagnostic yield gained from taking radiographs and the potential disadvantages of exposure to radiation.
2. If radiographs are to be taken, the clinician must choose films that will provide the highest diagnostic yield for the least possible radiation.
3. There should be a signed and dated radiographic report in the notes pertaining to all radiographs taken.
4. Should there be difficulty in interpretation of radiographs an opinion should be sought from a radiologist and a written report requested.

Choice of Techniques

Fast (E- or F-speed) films and narrowly collimated X-ray beams should be used to decrease radiation doses as much as possible. Three types of film are routinely used in conventional dental radiography: dental panoramic tomograms, bitewings and periapicals.

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1. 口腔全景 X 线断层片

口腔全景 X 线断层片给出了口腔一般的全面观察，对牙齿和颌骨进行普通检查，如图 9-1 所示。遗憾的是由于与颈椎重叠和该区域的扭曲变形，经常发现在水平面上的失真，特别是围绕下前牙区的 DPTs 非常不清晰。尽管多数临床医师认为 DPT 图像没有口内胶片的图像质量好，但近年来 DPTs 的质量已得到大的改进，而且拍摄DPT 的辐射剂量明显少于拍摄全口口内 X 线片。

Dental panoramic tomograms (DPT)

Dental panoramic tomograms give a general overall view of the mouth, permitting a general examination of the teeth and jaws. An example is shown in Fig 9-1. Unfortunately, distortions in the horizontal plane are frequently found. In particular, DPTs are less clear around the lower anterior region owing to both superimposition of the cervical spine and distortion in this area. The quality of DPTs has improved greatly in recent years, though most clinicians would accept that the image quality is not yet as good as that from intra-oral films. The radiation dose from a DPT is, however, significantly less than that from full-mouth intra-oral radiographs.

2. 殆翼片

殆翼片可在两个平面使用——垂直面和水平面。水平殆翼片（图 9-2）在一般临床实践中常用来发现邻面龋，也可用来检查轻度、中度牙周疾病边

Bitewings

Bitewings can be used in two planes – vertical or horizontal. Horizontal bitewings (Fig 9-2) are often taken in general practice to look for interproximal caries and can

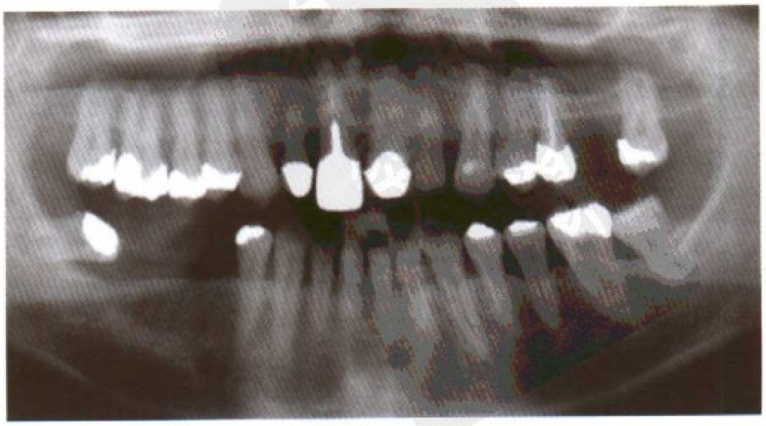


图 9-1　口腔全景 X 线片（DPT）

Fig 9-1 DPT radiograph.

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图 9-2　水平 X 线殆翼片

Fig 9-2 Horizontal bitewing radiographs.

缘骨丧失。通过此法，一张胶片就能显示上下牙弓的牙槽嵴而且辐射剂量相对较小。但是这种胶片不显示根尖周组织，有时也不能显示根分叉区。与水平殆翼片相比，垂直殆翼片尽管显示的牙齿较少，但显示的牙齿比例更大。垂直殆翼片尽管经常不能显示根尖周区，但可用来显示拍摄牙边缘骨嵴和根分叉区。

also be used to look for marginal bone loss in mild to moderate periodontal disease. In such circumstances, a single film will show the bone crest in both arches and the radiation dose is relatively small. This type of film does not, however, show the periapical tissues and sometimes the furcation areas are also not visible. Compared to horizontal bitewings, vertical bitewings show a greater percentage of the teeth being filmed though fewer teeth are displayed on each one. Vertical bitewings may be used to display the marginal bone crest and the furcation areas of the filmed teeth though often the periapical areas are not visible.

3. 根尖片

当需要观察根尖和根尖周组织最细微的细节时选择拍摄根尖片。从根尖片（PAs）可观察个别牙及其支持组织可能的最多的信息——包括根分叉和根尖周区——尽管与拍摄 DPT 相比拍摄全口根尖片的辐射剂量增大。由于使用了一种长锥体平行技术并使用胶片夹持器，所以能产生最少畸变失真，得到最佳结果。二等分角技术因其

Periapicals

Periapicals are the films of choice when the finest detail is required and imaging of the apical and periapical tissues is necessary. Periapical views (PAs) will give the most information possible about individual teeth and their support – including the furcation and periapical areas – though if full mouth films are taken there is an increased radiation dose for the patient compared to

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会低估骨丧失应该避免使用。如果临床医师希望估计牙根长度和形状应选择根尖片，图9-3显示的是一套全口牙齿根尖周片。为了达到最低的辐射剂量，在2年内重复拍摄全口根尖片的情况是少有的。但是如果在这段时间内出现特殊情况，也可能有必要重复个别牙的根尖片，但这样做必须有令人信服的临床理由。

taking a single DPT. The best results are obtained using a long cone paralleling technique with the use of film holders, as this produces the least distortion. The bisecting-angle technique should be avoided because it results in underestimation of bone loss. If the clinician wishes to assess the root length and shape then the PA is the film of choice. A set of full-mouth periapical films is shown in Fig 9-3. In order to minimise radiation dosage it would be unusual to repeat full-mouth periapicals within two years. It may, however, prove necessary to repeat individual periapical films within this time if particular problems arise, though there must be a compelling clinical reason to do so.

较新的数码成像技术越来越普及并表现出许多优点，并不只是因为它降低了患者的辐射。另外，它能对拍摄的图像进行多种处理，方便进行特殊检测的评估。数码减影成像术（DSR），特别在突出骨丧失中的小变化时非常

The newer technique of digital radiography is becoming more widely available and presents a number of advantages, not least because it reduces the radiation exposure for the patient. In addition, it permits varied manipulation of the images produced

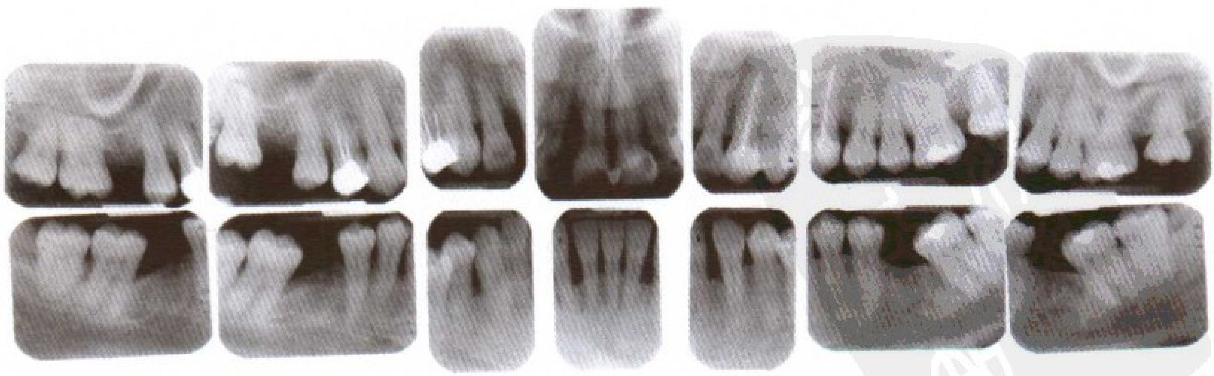


图9-3 全口根尖X线片

Fig 9-3 Full-mouth periapical radiographs.

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有用。使用该技术进行精确比较时，拍摄的图像必须精确地以相同的方法进行。为达到此目的要求使用胶片夹持器和殆垫等，同时也使得实际过程变得复杂而耗时，使用这种技术需要大量资金支出，但它们似乎在将来能展现其价值。

and facilitates the assessment of specific measurements. Digital subtraction radiography (DSR), in particular, is useful for highlighting small changes in bone loss over time. For accurate comparisons using this technique, it is essential that the films being examined have been taken in precisely the same way. The use of film holders and stents, etc. is required to achieve this, which inherently makes the physical process rather more complicated and time-consuming. Switching to this technology involves considerable capital outlay but it seems to be where the future lies.

采用何种胶片拍摄

没有固定的规则，表9-1是一个指导建议。

Which Films to Take

There are no fixed rules for this but Table 9-1 is a guide.

X线片的优点

X线片明显能使硬组织形象化，所以能在出放射学报告前对牙根和牙槽骨解剖及病理进行检查。

Advantages of Radiographs

Radiographs clearly facilitate visualisation of hard tissues, thereby enabling examination of root and bone anatomy and pathology, prior to preparation of the radiological report.

放射学报告

牙周组织变化的速度在临床上常常有重要意义，所以记录放射学发现很重要，它能够允许对病理特征进行长期纵向观察检测。准备报告时应考虑以下几点，阴性结果也应包括在内。

The Radiological Report

The rate of change is often significant so documentation of radiological findings is important as it permits longitudinal examination of features over time. When preparing the report there should be consideration of the following points. Negative findings

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表 9-1　采用何种胶片拍摄——建议

Table 9-1 Which films to take – a suggestion.

疾　病	分　布	严 重 程 度	X 线 片
龈　炎			无
牙周炎	广泛性	轻度（LOA ≤ 3 mm）	DPT
		中度（3 mm < LOA < 5 mm）	DPT 和局部区域的辅助 PAs 或垂直殆翼片（如根分叉病变）
		重度（LOA > 5 mm）	直立牙的PAs或DPT如果有间隙
牙周炎	局限性——只感染后牙	轻度	水平殆翼片
		中度	垂直殆翼片
		重度	感染牙PAs或初期拍DPT ± 辅助 PAs
牙周炎	局限性——只感染前牙	轻度	感染牙PAs或初期拍DPT ± 辅助 PAs
		中度	感染牙PAs或初期拍DPT ± 辅助 PAs
		重度	感染牙PAs或初期拍DPT ± 辅助 PAs
牙周炎	局限性——累及前牙和后牙	轻度	DPT
		中度	初期拍DPT ± 后牙垂直殆翼片 ± 前牙 PAs
		重度	直立牙PAs或初期DPT ± 辅助 PAs

1．用图表描述存在的牙齿。

2．骨吸收。在牙周诊断中尤其重要。但应该记住通过任何清晰

should also be included.

1. Charting of the teeth present.

2. Bone loss. Consideration of this is important in periodontal diagnosis. It

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度的X线片都只能观测邻间骨吸收情况，因为牙齿的高阻射性阻碍了对覆盖在牙上的骨组织的观测。应该考虑骨丧失的三个方面：严重程度、方式和分布。

should be remembered, however, that only interproximal bone can be examined with any clarity from radiographs, as the high radio-density of the teeth themselves prevents visualisation of bone overlying them. Three aspects of bone loss should be considered: severity, pattern and distribution.

严重程度

过去常常用从ACJ到牙槽骨嵴的毫米线性测量值来计算（图9-4）。但是由于许多原因，后来被认为是一个不合适的办法。首先它没有注意到牙齿的固有长度。当一个牙根长度很短时发生5mm的直线骨丧失要比一个具有更长牙根的人发生同样的骨丧失严重得多。其次，X线照片只是可能被缩短或延

The severity

In the past this used to be calculated as a simple linear measurement in millimetres (mm) from the ACJ to the bone crest (Fig 9-4). This was subsequently judged to be an inadequate method, for a number of reasons. First, it took no cognisance of the original root length. This is significant as a 5mm linear loss of bone in someone with short roots is much more serious

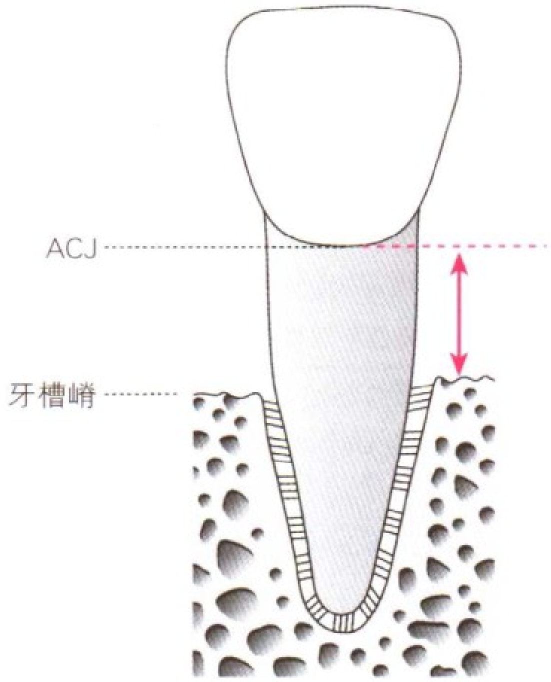


图9-4　ACJ到牙槽嵴的线性测量

Fig 9-4 Diagram to illustrate old-style linear measurement of bone loss from ACJ to bone crest.

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长的一颗或多颗牙齿的图像，所以从X线照片上直接进行测量同样可能失真变形，故最好认为任何获得的结果都是可疑的。解决的办法是观察检测没有骨覆盖的牙根比例，因为不管图像是缩短或延长，这个比例是不会改变的。测量骨丧失用公式表示为：

$$\frac{\text{ACJ} - \text{牙槽骨嵴(mm)}}{\text{ACJ} - \text{根尖(mm)}} \times 100\%$$

例如一个面可能显示50%的骨丧失。

方　式

两种主要的骨丧失方式是可以辨别的。一种是水平骨丧失（图9-5），它是在牙齿之间发生均匀的邻间牙槽嵴丧失。这种形式的骨丧失与骨上真性牙周袋相关，此时牙周袋底位于牙槽嵴的冠方（图9-6），另一种是垂直/角形骨丧失。"垂直"和"角形"可交换使用，它们的意思是一样的（图9-7），垂直骨丧

than the same loss occurring in someone with much longer roots. Secondly, it was recognised that a radiograph presents only an image of a tooth or teeth and that this image may be foreshortened or lengthened. Direct measurements taken from a radiograph may thus be similarly distorted, making any results gained questionable at best. The solution is to look at the percentage of the root no longer covered by bone, as this proportion will not change irrespective of foreshortening or lengthening. The measurement of bone loss is thus represented by the formula:

$$\frac{\text{ACJ} - \text{bone crest (in mm)}}{\text{ACJ} - \text{root apex (in mm)}} \times 100\%$$

Thus, for example, a surface may show 50% bone loss.

The pattern

Two main patterns of bone loss are discernible. The first is horizontal bone loss (Fig 9-5), which occurs when the interproximal bone crest is lost essentially evenly between the teeth being examined. This pattern of bone loss is associated with supra-bony true pockets, in which the base of the pocket lies coronal to the bone crest (Fig 9-6). The other is vertical/angu-

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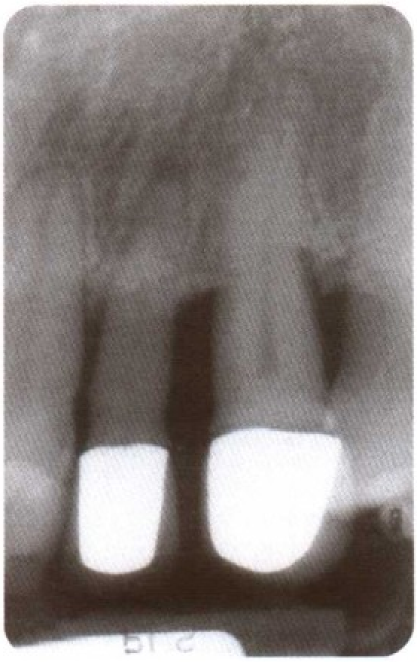


图 9-5　牙槽骨水平丧失

Fig 9-5 Radiograph demonstrating horizontal bone loss.

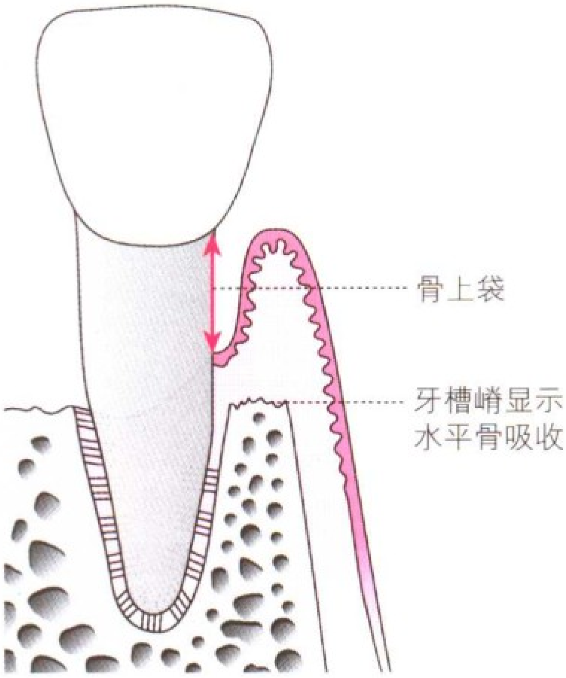


图 9-6　骨上袋

Fig 9-6 Diagram representing supra-bony pocketing.



图 9-7　垂直 / 角形骨丧失

Fig 9-7 Radiograph demonstrating vertical/angular bone loss.

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失出现在一侧邻间牙槽嵴的丧失大于另一侧时，这种形式与骨下真性牙周袋相关，此时牙周袋底位于形成的骨缺损内（图 9-8）。

lar bone loss. The terms "vertical" and "angular" are often used interchangeably and mean the same thing (Fig 9-7). Vertical bone loss occurs when more bone is lost on one side of the interdental bone crest than on the other. This pattern is associated with infra-bony true pockets, in which the base of the pocket lies within the bone defect created (Fig 9-8).

分　布

不管是局限性或广泛性，均应注意骨丧失的分布，如果≤30%的位点受到影响时称为局限性，如果受影响的位点>30%则认为是广泛性的（见第 6 章）。

The distribution

This should be noted as being either localised or generalised. One way of defining this would be to say that if ≤ 30% of sites are affected the condition would be described as being localised and if >30% of sites are affected it would be considered to be generalised (see Chapter 6).

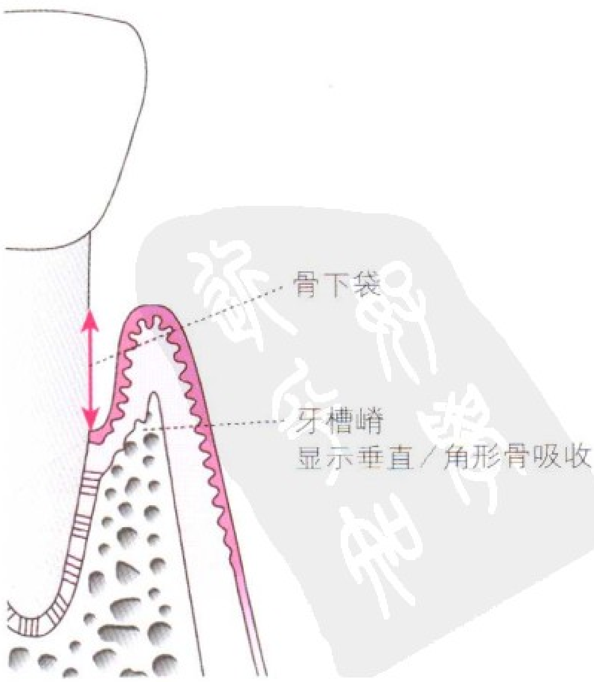


图 9-8　骨下袋

Fig 9-8　Diagram representing infra-bony pocketing.

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3. 龋。不管是原发性还是继发性龋都应引起重视，且修复措施应该成为随后治疗计划的一部分（图 9-9）。

4. 悬突修复体。请注意，对悬突修复体应及时去除或重新修复使其轮廓与牙体外形一致。它们是明显的菌斑滞留体，当它们存在时从事牙周治疗显得毫无意义，这对牙周疾病高易感者尤其如此。有时悬突可用抛光条、抛光磨石、低速手机（图 9-10）或使用超声洁治器去除，如果这些均无效时，则需要替换修复体。

3. Caries. This should be noted whether “primary” or “secondary” and corrective measures should be part of the subsequent treatment plan (Fig 9-9).

4. Overhanging restorations. These should be noted and their removal or recontouring should be quickly effected. They are obvious plaque traps and there is no point in embarking upon periodontal therapy if they remain. This is particularly true for patients with a high susceptibility to periodontal disease. Sometimes overhangs may be removed by use of finishing strips, finishing burs, reciprocating handpieces (Fig 9-10) or even via the use of an ultrasonic scaler. If none of these options proves effective, then replacement of the affected restorations is required.



图 9-9　第 1 和第 2 磨牙邻面龋和第 3 磨牙殆面龋

Fig 9-9 Radiograph demonstrating caries between the first and second molars and on the third molar.

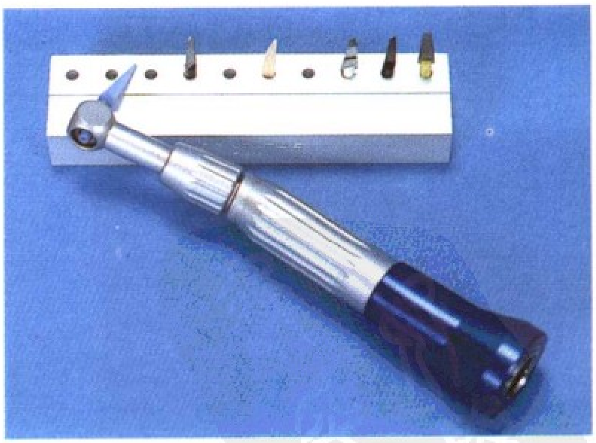


图 9-10　低速手机

Fig 9-10 Photograph of reciprocating handpiece with inserts.

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5. 牙石。X线片上是否显示牙石决定于两个因素:

钙化程度

牙石的钙化程度是不同的，在钙化最小阈值以下的牙石在X线片上将无法显示。

位 置

牙石位于邻面区域时通常才可见，由于牙齿的阻射性使得与它们重叠的牙石模糊不清。

由于牙石的存在，在解释X线片时必须谨慎，如果在胶片上看见牙石则它一定是存在的，如果没有看见，它也可能存在，但在其他位置或钙化特别低时可能不能观察到牙石（图9-11）

5. Calculus. Two factors determine whether calculus is seen on a radiograph.

Degree of mineralisation

The degree of mineralisation varies and there is a minimum threshold below which it will not be visible on radiographs.

Location

Calculus on the tooth will usually only be visible if it is located in the interproximal regions. This is because the teeth are so radio-dense that they obscure superimposed calculus.

The interpretation of radiographs with regard to calculus must therefore be carefully considered. If calculus is seen on a film it is definitely present. If it is not visible on a film then it may be present, but in another location, or may be too poorly mineralised to permit visualisation (Fig 9-11).

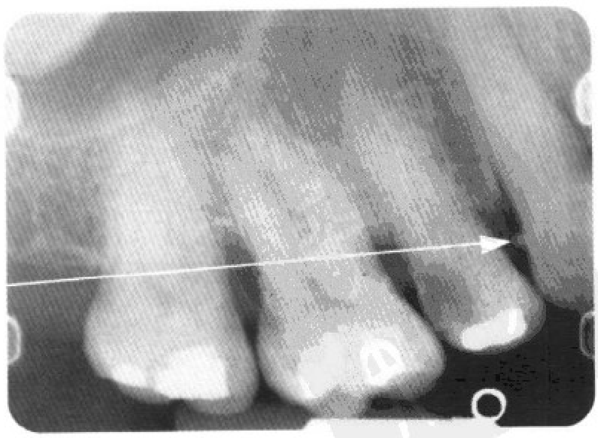


图 9-11 X线片示牙石

Fig 9-11 Radiograph demonstrating calculus.

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6. 根尖周病变。必须能够辨别根尖周病变，如果可能应与先前的胶片进行比较，以获得病变存在的时间长短和病变范围是否扩大或减小的提示。根尖周病变可能是在X线片上的一个偶然发现，或可能是为了证实临床检查的怀疑时发现（图9-12）。这种病损出现时的初始体征是硬骨板连续性丧失（图9-13），这时应该检查牙齿的活

6. Periapical pathology. This must be identified and should, if possible, be compared with previous films to provide an indication of how long a lesion has been present and whether it is enlarging or declining in size. Periapical areas may be an incidental finding on a radiograph or the film may confirm suspicions from the clinical examination (Fig 9-12). The first sign of this type of lesion occurring is loss of

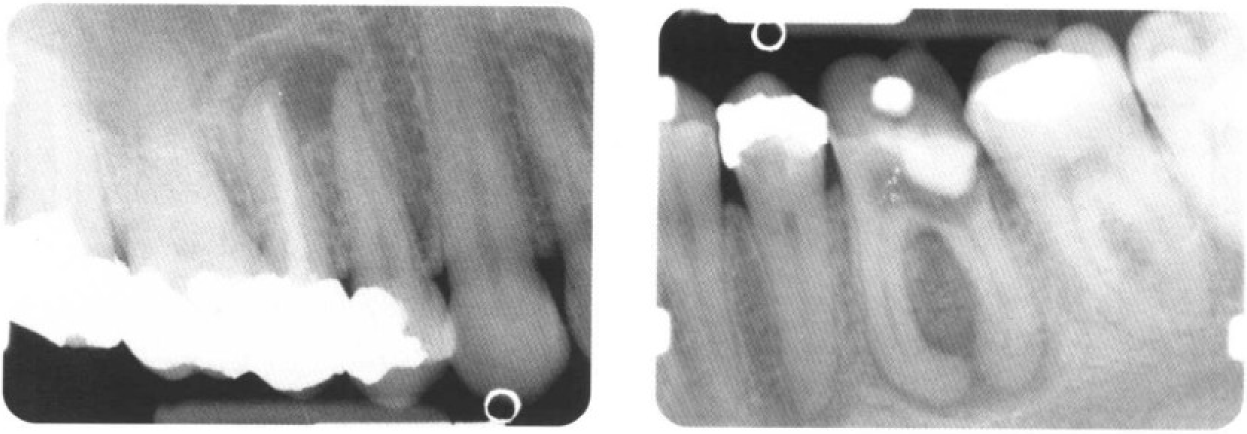


图9-12　X线片示根尖周区

Fig 9-12 Radiographs demonstrating periapical areas.

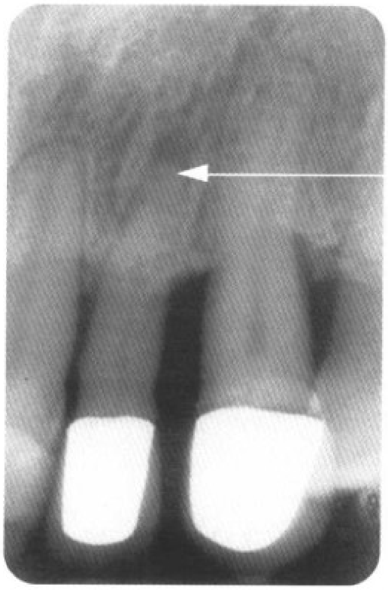


图9-13　X线片示硬骨板消失

Fig 9-13 Radiograph demonstrating loss of the lamina dura

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力，同样应该检查𬌗关系情况。如果一个牙齿牙周和牙髓均受感染——真正的联合病损——牙髓治疗应该先于牙周治疗，因为变性坏死的牙髓残留物会通过根尖孔、侧支根管和副根管，甚至可能从牙本质小管释放毒素。这些毒素进入牙周韧带后，即使经过牙周治疗，它们也会妨碍牙周位点的愈合。所以第一步是去除牙齿内毒素储库。同时重要的是不要将根尖周病变与正常解剖特征混淆。普遍存在的问题有颏孔与下颌前磨牙的重叠（图9-14），切牙孔与上颌中切牙的重叠（图9-15）以及上颌窦与上颌后牙重叠时显示根尖周病损的外观表

the continuity of the lamina dura (Fig 9-13), and if this is observed the tooth involved should have its vitality checked and the occlusion should be similarly examined. Should a tooth turn out to be both periodontally and endodontically involved – a true combined lesion – the endodontic treatment should always precede periodontal therapy. This is necessary as the degenerating remains of a necrotic pulp release toxins through the apex, through lateral and accessory canals and possibly through dentinal tubules. Such toxins entering into the periodontal ligament will prevent healing of the site even if periodontal therapy is undertaken. The first step is thus to remove



图9-14 X线片示下颌前磨牙的颏孔

Fig 9-14 Radiograph demonstrating the mental foramen lying close to the lower premolars.



图9-15 X线片示上中切牙顶部的切牙孔

Fig 9-15 Radiograph demonstrating the incisive foramen lying close to the apices of the upper central incisors.

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现（图9-16）。

the reservoir of toxins from within the tooth. It is also important not to confuse normal anatomical features with periapical disease. Commonly, problems arise with apparent superimposition of the mental foramen upon the lower premolars (Fig 9-14), the incisive foramen upon the upper central incisors (Fig 9-15) and the maxillary sinus upon the upper posterior teeth sometimes giving the appearance of periapical areas (Fig 9-16).

7. 根分叉病变的建议。根分叉病变的诊断是临床性的（见第8章），尽管X线照片上箭头所指的病损（图9-17）提醒临床医师有根分叉病变的可能，但要证实还需再次进行临床检查。

7. Suggestion of furcation involvement. The diagnosis of furcation involvement is always clinical (see Chapter 8), though the presence of an arrowhead lesion (Fig 9-17) on a radiograph should alert the clinician to the possibility and ensure that clinical re-examination occurs.



图9-16　X线片示上颌牙根与上颌窦重叠致根尖周区透明

Fig 9-16 Radiographs demonstrating apparent periapical areas due to superimposition of maxillary roots over antral air space.

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图 9-17　X 线片示箭头状病损

Fig 9-17 Radiograph demonstrating an arrowhead lesion.

8. 根折和其他病理变化。X线照片可以证实根折的存在。X线片上的任何显示都应该重视，如果有必要应征求专家意见作出诊断。

8. Presence of root fractures and other pathology. Radiographs may demonstrate the presence of a root fracture. Any present should be noted and if necessary expert advice sought for specific diagnosis.

X 线片的缺点

要仔细观察胶片并作出正确的解释，必须认识X线片的缺点。它们包括：

Disadvantages of Radiographs

The disadvantages of radiographs must be understood if correct interpretation of the scrutinised films is to occur. These include:

1. 重叠。一张 X 线照片将三维物体转变为二维图像将不可避免地出现重叠现象。包括三种情况：牙与牙的重叠，牙与骨的重叠以及骨与骨的重叠。

牙与牙重叠（图9-18）　这意味着不可能涉及牙齿的所有的面，因为它们的图像至少部分是互相覆盖的。

1. Superimposition. A radiograph converts a three-dimensional object into a two-dimensional image and this inevitably results in superimposition. This occurs in three ways: tooth-on-tooth superimposition, tooth-on-bone superimposition and bone-on-bone superimposition.

Tooth-on-tooth superimposition (Fig 9-18). This means that it is not possible to look at all surfaces of the teeth involved, as their images lie at least partially on top of one another.

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牙与骨重叠 在全口可见。牙槽骨只在邻面可见的原因是因为牙齿是高度X线阻射的，使重叠在牙面的骨组织不能清楚显示。

骨与骨重叠（图9-19） 由于牙槽骨颊、舌侧骨板的吸收不一定是均匀的，当发生重叠时可看见两个骨影。另外，如果骨皮质是完整的，这些骨壁内的缺陷程度在X线片上可能被颊、舌侧骨板掩盖。但如果临床医师非常仔细地检查这些位点，

Tooth-on-bone superimposition occurs all around the mouth. This is the reason that bone is visible only interproximally as the teeth being so radiodense do not permit clear visualisation of the bone overlying them.

Bone-on-bone superimposition (Fig 9-19) occurs because the buccal and lingual plates of bone do not necessarily resorb evenly and when superimposed a double shadow of bone may be seen. In addition, if cortical bone remains intact the extent of a defect lying within these walls may be

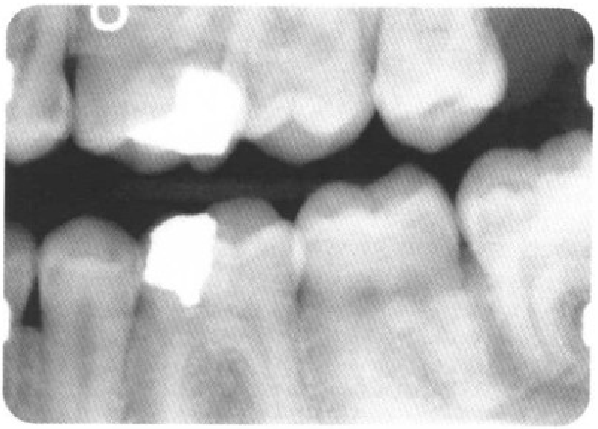


图9-18 X线片示牙与牙重叠

Fig 9-18 Radiograph demonstrating tooth-on-tooth superimposition.

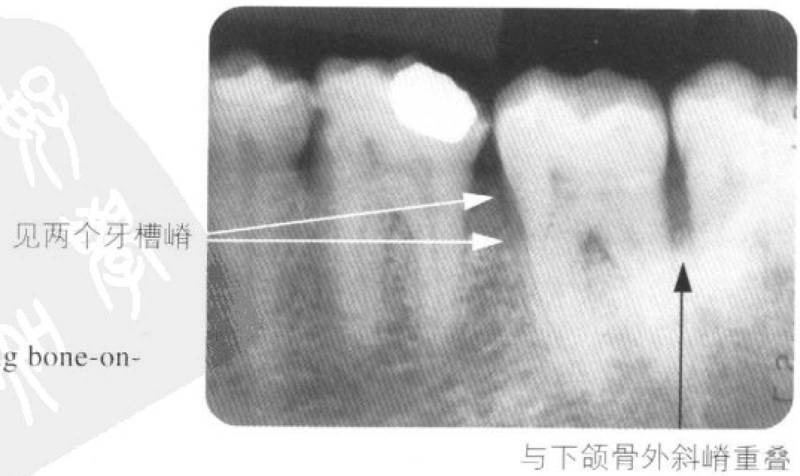


图9-19 X线片示骨与骨重叠

Fig 9-19 Radiograph demonstrating bone-on-bone superimposition.

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仍可能看见骨密度的变化。

2. 过低估计骨丧失。如果比较一个位点的X线片和同一位点切开暴露的情况，X线照片常常显示较低的骨丧失。当决定是否保留或治疗重度牙周疾病的牙齿时记住这一点非常重要。

3. 没有疾病活跃性的指征。轻探牙周袋底无出血预示疾病无活跃性。X线片没有表明这一临床体征，因为X线片指示的只是硬组织的历史疾病经历。

4. 没有表明牙周袋深度、附着丧失或牙齿松动度。所有这些都得通过临床进行评估，X线片不起作用。

5. 有限地说明疾病的历史。X线片是拍片时硬组织的一个快照图像，所以不可能通过观察一套X线片知道疾病发展的时间长短或疾病发生次数。也就是说不可能确定X线片上看见的病损是否是一段时间前发生的病损以及疾病现在是否已经停滞，

masked radiographically by the intact buccal or lingual bone plates. However, changes in bone density may be seen in such sites if the clinician examines the sites very carefully.

2. Underestimation of bone loss. This means that if a radiograph of a site is compared with surgical exposure of the same site, the radiograph would usually underestimate the bone loss. It is important to remember this when deciding whether to retain and attempt to treat teeth with severe periodontal involvement.

3. No indication of disease activity. Lack of disease activity is indicated by a lack of bleeding from the base of the pockets after gentle probing. Radiographs give no indication of this clinical sign as they indicate only historical disease experience in hard tissues.

4. No indication of pocket depth, loss of attachment or mobility. All these are assessed clinically and radiographs have no role in this process.

5. Limited indication of the history of the disease. Radiographs give a snapshot image of the hard tissues at the time the films were taken. It is thus not possible by looking at one set of films to know either for how long any disease has been progressing or how many episodes of disease have taken place.

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或者它是否全部是新近发生的。

6．随时间进展发生的小变化的评估是很困难的。即使采用连续的常规X线片进行评估来检测骨丧失的微小变化也是很困难的。这个问题通过数码减数成像技术至少可以部分解决。

7．患者的辐射。辐射是患者为诊断付出的生理代价，所以必须只在有明确的临床指征时才拍摄X线片。在获取需要的信息时辐射剂量必须是尽可能最小，且必须对X线片作出全面报告以获得最多的信息。

有这么多缺点，X线片是一个好的检测方法吗？

答案是有条件的肯定，尽管临床医师有责任非常仔细地估量X线片的必要性和需要拍摄X线片的类型和数量。

特殊检测

相对来说很少患者需要进行特殊检测，但对有的患者是必要的。

This means that it is not possible to ascertain whether destruction seen on a film took place some time ago and the disease has now "burnt out", or whether it all happened recently.

6. Assessment of small changes over time is difficult. Even with sequential conventional radiographic assessment, detecting small changes in bone loss is difficult. This problem may be at least partly overcome by use of DSR.

7. Radiation of the patient. Radiation is the biological cost, paid by the patient, for the diagnostic yield that the films provide. It is because of this that films must only be taken when there is a clear clinical indication to do so. The radiation dose used must be the minimum possible to obtain the information needed and the films taken must be fully reported to maximise the information gain.

With So Many Disadvantages Are Radiographs a Good Thing?

The answer to this is a qualified yes, though the clinician has a duty to weigh up both the need for radiographs and the numbers and types required very carefully.

Special Tests

Special tests are required by relatively few patients but are essential for some. The

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最常见的特殊检测有：

1. 血液检测。为调查生物学价值的一个特殊部分，有许多不同类型的血液检测，对疾病与菌斑水平不一致的重度牙周病患者应该考虑进行血液检测。尽管经指导有明显合作的证据但治疗的反应很差时血液检测显得尤其重要。如果牙周病患者有系统性疾病时也应考虑进行血液筛查，其表现的牙周破坏，很可能至少部分可能是由系统性因素所致。最常见的血液检测详细说明如下：

全血细胞计数（FBC）——其提供了关于血细胞、血小板数和它们的相关水平的信息，可发现例如中性粒细胞减少症，一种据报道与牙龈溃疡和高危牙周破坏相关的疾病。

分类白细胞计数——常规通过FBC得到，能够帮助排除疾病如白血病。白细胞增多与重要牙周炎相关，因为人体对相关病原体发生免疫反应。

most common are:

1. Blood tests. There are many different types of blood test, designed to investigate a particular aspect of biological importance. Blood tests should be considered in patients with severe disease which is not commensurate with their plaque levels. This becomes even more important if the response to treatment is poor despite clear evidence of co-operation with instructions given. Blood screening should also be considered if patients with periodontal disease are systemically unwell. It is possible that the periodontal destruction exhibited may be accounted for, at least in part, by systemic factors. The most common blood tests are detailed below.

The full blood count (FBC) – this provides basic information on blood cell and platelet numbers and their relative levels. This may highlight, for example, the presence of a neutropenia, a condition reported to be associated with gingival ulceration and increased risk of periodontal damage.

A differential white blood cell count– this is obtained routinely from a FBC, and can help to eliminate conditions such as leukaemia. Increased leucocyte numbers (leucocytosis) are associated with significant periodontitis, as the

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body mounts an immune response to the relevant pathogens.

血涂片——血涂片是检测血细胞大小和形状。FBCs 检测结果能提供细胞尺寸增加或减少的信息（大／小红细胞增多），就像在维生素 B_{12}／叶酸缺乏和铁离子缺乏时所见一样。但是细胞形态的细微改变需要血液学专家在显微镜下观察，而不是依赖FBCs的自动血细胞计数检测（血球计数器）。例如通过FBC不能发现血小板功能缺陷，因为血小板数目可能是正常的。

Blood films – are tests for blood cell size and shape. Results from FBCs will provide information on increased or decreased cell size (macro-/micro-cytosis) as seen in B12/folate and iron deficiency respectively. However, subtle changes of cell form require a haematologist to view cells microscopically, rather than relying on automated cell counters (haemocytometers) for FBCs. Defects of platelet function, for example, will not be detected by a FBC, as platelet numbers may be normal.

血糖检测——如果怀疑有糖尿病时可检测血糖，如果结果不清楚，则需要检测糖基血红蛋白水平（见第 4 章）。

Blood glucose testing – is performed if diabetes is suspected, though if the results are unclear, glycated haemoglobin levels may be required (see Chapter 4).

免疫学——不同水平的免疫球蛋白和补体，与所谓的急性期蛋白（肝脏产生）一样，对感染性负荷不能提供特异性指征。但是对脱屑性龈炎的研究则依赖使用间接荧光检测法来识别对上皮基底膜或细胞间结合剂（钙依赖蛋白分子）的自动抗体。PMNL 功能的检测也可帮助识别侵袭性牙周炎潜在的中性粒细胞功能缺陷，但是大多数免疫学鉴定分析最好在专家中

Immunology – Various levels of immunoglobulin and complement, as well as so-called acute-phase proteins (produced by the liver), provide non-specific indications of infective load. However, the investigation of desquamative gingivitis relies upon identifying auto-antibodies to epithelial basement membrane or intercellular cement (cadherin molecules), using indirect immunofluorescence. PMNL-function tests may also help identify

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心进行。

underlying defects of neutrophil function in aggressive periodontitis, but most immunological assays are best performed in specialist centres.

2. 活力测试。如果一颗牙有活力丧失的可能，则有必要进行活力测试。如果牙齿颜色变暗，在X线片上会有明显的根尖周透射影像，或有临床表现，包括根分叉病变存在时，则可能出现活力问题，否则建议进行活力测试。明白活力是否丧失是很重要的，因为其结果将必然改变治疗计划，包括是进行根管治疗还是拔除感染牙。因为常常出现假阳性和假阴性结果，所以活力测试应该使用至少两种方法——通常采用氯乙烷和牙髓电测仪，尤其在老年患者和大面积修复的牙齿上这种情况常常发生，因为产生大量的继发性牙本质。测试时应将对侧牙和邻牙常规作为对照进行测试。如果活力测试结果不明确，则制备一个测试窝洞进行最后的酸性试验。如果牙齿仍有活力，当用牙钻在釉牙本质界处钻磨时患者会有反应，随后应在窝洞中充填小的修复体。如果患者没有反应，术者能确定牙齿已无活力可继续进行根管治疗或拔除患牙。个别位点对牙周治疗没有反应的常见原

2. Vitality testing. This is essential if there is a possibility that vitality may have been lost in a tooth. The question may arise because a tooth becomes darkened, because evidence of periapical radiolucency is seen on radiographs, or because the clinical presentation, including the presence of furcation involvement, otherwise suggests it. It is important to know if loss of vitality has occurred because the outcome will inevitably change a treatment plan to include either root canal treatment or extraction of the affected tooth. Testing should be done using at least two methods – commonly application of ethyl chloride and an electric pulp tester – as false positives and negatives are common. This is true especially in elderly patients whose teeth have large amounts of secondary dentine and for a similar reason in heavily restored teeth. The contra-lateral and adjacent teeth should also be routinely tested as controls. If results of vitality testing are equivocal, preparation of a test cavity is ultimately the acid test. If the tooth is still vital the patient will respond as the amelo-dentinal junction

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因是牙髓源性的微生物再感染牙周袋。

of the tooth is broached by the bur and a small restoration should subsequently be placed. If the patient does not respond, the operator can be confident that the tooth is non-vital and progress to root canal therapy or extraction. A common reason for individual sites being unresponsive to periodontal therapy is due to pocket reinfection from organisms of endodontic origin.

3. 使用牙胶尖逆向追踪感染源。当患者出现瘘管时，由于炎症会沿着阻力小的途径扩散，所以总是不能直接判断其来源。如果存在广泛性牙周疾病和牙列经过大量修复时，识别感染来源显得尤其困难。但是要进行适当的治疗，隔绝病因是基本前提。一个实用的方法是在瘘管中放入一根牙胶尖，轻轻将牙胶尖沿瘘道逆向探入，直至有阻力感，此时如果拍摄X线片，就能发现感染源常常位于牙胶尖所在的位置（图 9-20）

3. Use of a gutta percha point to track back to the source of infection. When a patient presents with a sinus, it is not always straightforward to decide upon the source of the problem, as infection will track along the path of least resistance. Identification of the source is especially difficult if there is generalised periodontal disease and the dentition is heavily restored. However, isolation of the cause is an essential prerequisite if appropriate therapy is to begin. One useful method is to introduce a gutta percha point into the sinus and gently explore it back along the sinus tract until resistance is felt. If a radiograph is taken at this time with the point in place, the source of the infection is often located at the site at which the point lies (Fig 9-20).

4. 研究模型的使用。对于伴有船关系问题，特别是有牙齿移位

4. Use of study models. These are useful in cases with occlusal problems and

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图 9-20　牙胶尖定位感染源

Fig 9-20 Using a GP point to locate the source of infection.







的病例是有帮助的。只有一系列一定时间间隔重复采取的模型才有价值，而不是指孤立的模型。模型展现经过一段时间后的变化，仅进行临床评估则会遗漏小而连续发生的变化。将一个早期的模型与一个系列模型中较晚期的模型进行比较会非常清楚地显示这一变化，并能提醒医师可能需要积极的治疗而不只是对一种状况可能需要的监视。研究模型在制定治疗计划时同样有用，晚期模型还可用来检验治疗进展，可用作维持治疗期的基线。

particularly in those demonstrating tooth migration. They are valuable not just singly but as a series, where models are produced at repeated intervals. Models display changes over a period of time and if the changes are small but continuous they could be missed if clinical evaluation alone was used. Comparison of an early model with one from later in the series often shows such changes very clearly and is useful in alerting the clinician to the prospect that active treatment rather than just monitoring of a situation may be required. The models are also useful in treatment planning and later may be

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used to check the progress of treatment and to act as a baseline for maintenance regimes.

5．照片的使用。照相记录不论对它自己还是作为研究模型的补充，常常是非常有用的。另一方面，一系列的照片对书面描述是一个巨大的补充，提供了有价值的基础信息并展示了疾病的进展情况，特别是软组织的病损情况。

5. Use of photographs. A photographic record is often extremely useful either on its own or as an addition to study models. Again, a series of photographs is a great addition to the written description and this record provides valuable baseline information and shows progress over time, particularly for soft tissue lesions.

6．牙龈活组织检查。尽管由于牙龈炎症无所不在，使得对牙龈组织的组织学描述更加困难，但对部分病例为了确定诊断，活检还是有用的。由于牙龈外形的任何缺陷都是不美观的而且可能促进菌斑积聚，所以活检选择的位置和使用技术也应该仔细考虑。当怀疑病损是恶性时，必须采用切取活检，因为在确定诊断后，切取位置和界标仍然存在，有利于更宽的切除。切除活检适用于明显是良性的病损。

6. Gingival biopsy. This is useful in some cases to confirm a diagnosis although interpretation of the tissue histology produced is often made more difficult by the omnipresence of gingival inflammation. The biopsy site chosen and the technique used should also be carefully considered as any defect in the gingival contour is extremely unaesthetic and may promote plaque accumulation. When there is suspicion that the lesion may be malignant, an incisional biopsy is necessary, so that if the lesion is confirmed as such, the original position and landmarks are still present for wider excision. Excisional biopsies are indicated for clearly benign lesions.

7．预测牙周疾病的测试。实质上所有临床牙周测试的问题在本质上都是回顾性的——因为它

7. Tests to predict periodontal disease. The problem with virtually all clinical periodontal tests is that they are retro-

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们证实疾病已经发生并帮助调查者衡量疾病发生的程度，但没有提供关于当前疾病活跃性的信息。最终希望是开发一种便宜、简单而快速的检测试验，能可靠地预测患者未来患病的危险。在20世纪80年代和90年代，许多研究集中在检测GCF中当前或未来疾病活跃性的标志上，而且许多试验获得对未来疾病活跃性70%～80%的预测性，这些试验由于椅旁应用麻烦且不实用而没有启动。理论上，预测试验允许集中珍贵稀有的资源给危险个体，希望初期筛查能够在早期识别疾病，阻止或至少将疾病进展降到最小。这将使治疗需求简单，从而提高治疗成功的机会。

spective in nature – that is they confirm that disease has taken place and help the investigator measure how much has occurred, but do not provide information on current disease activity. The ultimate hope is for the development of a test which is cheap, simple and quick to perform, that will reliably predict patients at risk of future disease. Many studies during the 1980s and 1990s focused on markers of current or future disease activity measured within GCF. Whilst many achieved 70%-80% predictivity for future disease activity, such tests were cumbersome and impractical for chair-side use and have never taken off. Theoretically, predictive tests would allow targeting of scarce resources at risk subjects such that initial screening would, one hopes, identify disease at an early stage and prevent or at least minimise disease progression. This would keep treatment needs simple and would thus increase the chance of the therapy being successful.

许多研究领域已经或现在正在被调查研究。近年来研究重点已经转移到遗传试验上，在白细胞介素-1 基因簇中几种基因多态性已被描述（见第 3、4 章），且已有报道部分基因多态性与几种慢性炎症性疾病包括牙周炎之间有相关性，现在能通过指血检测来

Many areas of research have been and currently are being investigated. The emphasis recently has moved in the direction of genetic testing. Several genetic polymorphisms have been described in the Interleukin-1 gene cluster (see Chapters 3 and 4) and associations have been reported be-

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检测这些基因多态性，但这种检测的应用限制在40～60岁的非吸烟牙周疾病患者，而且只能在这一人群范围进行，它对所有的英国人群则无效。

由于牙周疾病的多因素本质，遗传试验似乎不可能是牙周炎的完美预示因子。遗传试验诱人的地方是它是一个终生惟一试验，能够真正促进正确的预防治疗程序的建立。总的来说遗传试验有许多潜在缺陷，在我们试图将遗传标记与不同牙周疾病联系起来之前，我们需要对是什么构成一种独特疾病的意见一致。除非我们能约定一种疾病明确的临床标准（分类），否则我们就不能调查研究何种基因或基因多态性与哪种疾病相关。

tween some of these and increased severity of several chronic inflammatory diseases including periodontitis. A peripheral blood "finger prick" test designed to detect these polymorphisms is now available, but its utility appears limited to predicting periodontal disease in non-smokers aged between 40 and 60 years of age, and only within certain demographic areas. It does not appear effective for all UK populations.

It seems unlikely that genetic tests will be perfect predictors for periodontitis, owing to the multi-factorial nature of the periodontal diseases. The attraction of genetic testing is that it is a once-in-a-lifetime test and would genuinely facilitate the establishment of truly preventive management programmes. There are many potential pitfalls with genetic testing as a whole and before we try to associate genetic markers with different periodontal diseases we need to be in agreement about what constitutes a particular disease. Unless we can agree clear clinical criteria (classifications) for a disease, we cannot investigate which genes, or polymorphisms of those genes, are linked to that disease.

患者的转诊介绍

如果因为临床问题的罕见或困难，和（或）患者患有相关的内科疾病而导致病例复杂时，总是应该考虑向专家请教咨询。但是判定哪些病例需要恰

Referral of Patients

Referral to a specialist should always be considered if a case is complicated because the clinical problem is unusual or difficult and/or the patient has a relevant medical

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当的咨询是困难的，读者应该查阅由英国牙周病学会于2000年10月发行的《转诊政策和护理参数》(www.bsperio.org)。

condition. Deciding which cases to refer appropriately can be difficult and the reader should consult "Referral Policy and Parameters of Care" published by the British Society of Periodontology in October 2000 (www.bsperio.org).

放射学的重要变化

在20世纪90年代中期，国家放射学防护委员会（NRPB）发表了一份关于妊娠期电离辐射使用的声明，可概括为牙科X线照相的正常选择标准不必受妊娠期女性患者在任何妊娠阶段可能发生的情况影响。另外，没有正当理由对拍牙片的患者常规使用铅围裙——实际上在拍全景X线片时肯定是不鼓励使用的。这方面和相关问题的更多的信息从NRPB文件“放射学家皇家学院和国家放射学防护委员会的关于基本牙科护理放射学标准指南的报告”（1990年发表）和“牙科医师安全使用X线设备指南”（2001年发表）上可获得（www.nrpb.org），这些问题另外的讨论在即将发表的“口腔临床要点快速掌握系列”中的《21世纪影像学》中可找到。

Important Changes in Radiology

In the mid-1990s, the National Radiology Protection Board (NRPB) issued a statement on the use of ionising radiation during pregnancy. It may be concluded from this that the normal selection criteria for dental radiography do not need to be influenced by the possibility of a female patient being at any stage of pregnancy. In addition, there is no justification for the routine use of lead aprons for patients undergoing dental radiography - in fact their use during panoramic radiography is positively discouraged. Further information on these and related matters is available from the NRPB documents "Guidelines on Radiology Standards for Primary Dental Care Report by the Royal College of Radiologists and the National Radiological Protection Board" published in 1990 and "Guidance Notes for Dental Practitioners on the Safe Use of X-ray Equipment" published in 2001 (www.nrpb.org). Additional discussion of these issues will be found in a forthcoming book in the *Quintessential series, 21st-century Imaging*.

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拓展阅读
Further Reading

1 Eley BM, Cox SW. Advances in periodontal diagnosis 1. Traditional clinical methods of diagnosis. Br Dent J, 184;1998:12–16

2 Eley BM, Cox SW. Advances in periodontal diagnosis 2. New clinical methods of diagnosis. Br Dent J, 184;1998:71–74

3 Guidelines on Radiology Standards for Primary Dental Care Report by the Royal College of Radiologists and the National Radiological Protection Board. National Radiology Protection Board, 2001

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5 Referral Policy and Parameters of Care. British Society of Periodontology, 2000 (www.bsperio.org)

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第 10 章 牙周诊断和预后

Periodontal Diagnosis and Prognosis

目　的

本章的目的是将前面的章节集中在一起，使临床医师能得出实际诊断并依此制定恰当的治疗计划。(更多的论述见《为牙周小组制定治疗计划》)

Aim

This chapter aims to draw together the preceding text to enable practitioners to arrive at a practical working diagnosis upon which to develop appropriate treatment plans. (For further discussion of this see *Quintessentials: Treatment Planning for the Periodontal Team*.)

要　点

阅读本章后读者能够自信地诊断大多数常见牙周疾病，并能特异性地针对个体患者的诊断添加细节。读者也能在个体患者基础上，识别局部和系统危险因素以及形成关于未来疾病进展危险程度的意见。

Outcome

Having read this chapter the reader should be able confidently to diagnose the majority of the more common periodontal diseases and to add detail to that diagnosis specific to the individual patient. The reader should also be able, on an individual patient basis, to identify local and systemic risk factors and to form an opinion about the degree of risk of future disease progression.

诊　断

临床上应该常规得出诊断并写入有签名和注明日期的病历记录。这应

Diagnosis

Reaching a diagnosis and entering it in signed and dated patients' records should

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该在患者第一次治疗完成检查后执行，并且在随后的维持期再评估就诊时应该重复进行。

be routine practice. This should occur after the examination is completed when the patient first attends for treatment and should then be repeated at all subsequent re-evaluation appointments during maintenance.

诊断应该是特定时间对特定口腔中出现的特定疾病的精确描述。这对于它自身和一段时间后的比较来说都是有用的。一段时间后疾病严重程度和分布的变化，就像见到的变化程度一样常常以给出变化速度指征的方式被注意。对于一个患者未来牙周疾病情况最好的指示器是过去的疾病经历，所以这个信息是非常有价值的。

A diagnosis should be a concise description of the presenting disease in a particular mouth at a particular time. It is useful both on its own and in a comparative way over a period of time. Changes in severity and distribution of disease over time are often highlighted in this way giving an indication of rate of change as well as just the degree of change seen. Since the best indicator for future periodontal disease in a patient is their past disease experience, this information is very valuable.

可能的牙周诊断

过去，人们谈论牙周疾病时倾向于它像是一个疾病实体。但多年后，已经认识到存在许多不同形式的牙周疾病，它们恰好影响同一组织。这些能用许多方法进行鉴别，包括患者的年龄、严重程度、类型、可见的病损分布以及系统性疾病的存在。在第6章中已经详细讨论了牙周疾病的分类，但在考虑菌斑引起的炎症性牙周疾病时，出现三种主要的诊断情况：

Possible Periodontal Diagnoses

In the past, people tended to talk about periodontal disease as though it was just one disease entity. Over the years, however, there has been recognition that there are many different forms of periodontal disease, which just happen to affect the same tissues. These can be differentiated in a number of ways including the ages of the patients involved, the severity, type and distribution of destruction seen and the presence of systemic involvement. Classification of periodontal diseases is discussed in detail in Chapter 6, but when considering plaque-induced inflammatory periodon-

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tal diseases three main diagnostic situations arise:

1. 稳定的牙周膜。这个诊断与临床健康状态是一致的，如果所有的牙周袋都比较浅时患者能够自己保持清洁（平均牙周袋深度常常≤3 mm），轻探牙周袋底时无出血。这是一个理想情况，因为：
   - 没有我们知道的常常先于或伴随牙周破坏的炎症证据。
   - 患者能保持较低的菌斑指数——希望低于他们自己个体疾病阈值。

如果患者出现这种情况则不需治疗，而且有理由认为他们对牙周疾病的易感性相对较低。但是，有时高易感性患者经过成功的牙周治疗后在维持治疗期也可表现如此。众所周知对于任何患者的牙周治疗都不能改变其固有的牙周疾病的易感性，但经过适当的治疗和患者良好的合作，疾病可以被控制。在这些病例呈现一个稳定的牙周膜是可喜的发现，但必须认识到疾病并没有治愈，还需要制定适当的个体维持治疗

1. A stable periodontium. This diagnosis is consistent with clinical health and is made if all the pockets are shallow enough to be cleaned by the patients themselves (usually taken to mean pocket depths of ≤3mm) *and* there is no bleeding from the base of the pockets upon gentle probing. This is the ideal situation since:
   - there is no evidence of the inflammation which we know often precedes and accompanies periodontal breakdown
   - the patient should be able to maintain low plaque scores – it is to be hoped below their own individual disease threshold.

If a patient presents in this condition no treatment is required and it is reasonable to conclude that their susceptibility to periodontal disease is probably relatively low. Sometimes, however, patients with higher susceptibility who have undergone successful periodontal therapy may also present in this way during periodontal maintenance. It is known that periodontal treatment does not change the innate susceptibility to periodontal disease of any patient. However, with adequate therapy and good co-opera-

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程序来继续防范。

tion the disease may be controlled. In such cases presentation with a stable periodontium is a welcome finding, but there must be recognition that the disease is never cured and continued vigilance in the form of an appropriate individually designed maintenance programme is required.

2. 牙龈炎。没有出现附着丧失（所以任何牙周袋肯定都是假性牙周袋），但有牙龈充血发红、牙龈肿胀和轻探时牙周袋底探诊出血。就像第 5 章中讨论的一样，我们知道牙龈炎是牙周疾病的第一阶段而且有些患者不会继续发展，不幸的是其他具有牙周疾病高易感性的患者牙龈炎进展迅速，并很快发展为牙周炎。为了更清楚地描述龈炎的诊断，有两个主要描述词应该采用：

2. Gingivitis. This term means that there is no loss of attachment present (hence any pockets present must be false pockets), but there is redness, swelling and bleeding on probing from the base of the pockets, following gentle probing. As discussed in Chapter 5, it is known that gingivitis is the first stage of periodontal disease and that some patients never progress beyond this. Unfortunately, other patients with higher susceptibility to periodontal disease progress rapidly through gingivitis and quickly develop periodontitis. There are two main descriptors which should be applied to gingivitis to provide a clearer diagnostic picture. These are:

• 急性或慢性。关于这些术语有很多混淆。以前常常认为“急性”就是指被描述的疾病状况本质是严重的而“慢性”则指较低的严重程度。但正确地说它们描述的是疾病发生的时间长短，急性是指一

• Whether the condition is *acute* or *chronic*. There is a great deal of confusion about these terms. It has often been assumed that “acute” means that the condition being described is severe in nature while “chronic” implies lower severity.

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个短时期，慢性则指一个较长的持续期（见第 6 章）。毫无疑问术语学能使人迷惑，但在牙周术语中龈炎的急性形式可包括如NUG、原发性疱疹性龈口炎或牙周脓肿。但是，如前面所论述的一样，这些疾病病例，比较少见，也遵循一个慢性过程，但可能都属于慢性龈炎类型中的一种。

The terms correctly, however, describe the length of time for which the condition has been present, with acute meaning for a short time and chronic describing a greater duration (see Chapter 6). There is no doubt that the terminology can be confusing, but in periodontal terms acute forms of gingivitis may include conditions such as NUG, primary herpetic gingivostomatitis or lateral periodontal abscesses. However, as previously discussed, such conditions may, more rarely, also follow a chronic course and the majority of gingivitis cases fall into one of the chronic gingivitis categories.

- 局限性或广泛性。两者间没有严格和可靠的鉴别标准，但 1999 年世界牙周疾病分类专题研讨会提出口腔中 30% 以内的位点受到影响时为局限性的，大于这个数值时为广泛性，局限性疾病的描述可包括受累牙齿和牙面。而广泛性疾病该补充则没有必要。

- Whether the condition is *localised* or *generalised*. There are no hard and fast rules about the differentiation between the two but the 1999 World Workshop on Periodontal Disease Classification suggests that the term "localised" be used if up to 30% of the mouth is affected and "generalised" if more than this figure is involved. A description of the affected teeth and surfaces involved in the localised form of the condition may be included. This addition is not necessary when the condition is generalised.

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举一个诊断的例子如"局限性慢性牙龈炎（累及下前牙和尖牙的所有牙面）"。

3. 牙周炎。牙周炎是指有附着丧失并导致真性牙周袋，轻探牙周袋底出血。出现附着丧失即牙周破坏，尽管不是说疾病不能趋于稳定，但从定义上讲是不可逆的。这种情况有三个描述能够最大程度地、清晰地描述牙周炎诊断。

- *发生的是何种类型的牙周炎。*第 6 章讨论了牙周疾病的分类系统，其描述能够使医师判定发生的是何种形式的牙周炎。绝大多数来就诊的患者都有慢性牙周炎，甚至侵袭性牙周炎也是慢性疾病，而不是急性。

- *疾病是局限性还是广泛性。*30%的衡量标准是一个指导，但却有点武断，所以应用时建议根据一般感觉。

- *疾病的严重程度。*关于疾病是

An example of a diagnosis would thus be "localised chronic gingivitis (affecting all surfaces of the lower incisors and canines)".

3. Periodontitis. This term is applied when there is loss of attachment resulting in the presence of *true* pockets *and* bleeding from the base of the pockets following gentle probing. The loss of attachment suffered means that the periodontal damage is, by definition, irreversible, though this does not mean that the condition cannot be stabilised. In this case three descriptors can be applied to the term periodontitis to maximise the clarity of the diagnosis.

- *Which type of periodontitis is present.* The classification system for this is discussed in Chapter 6 and the descriptions found there should enable the clinician to decide which form of periodontitis is present. The vast majority of patients presenting for treatment have chronic periodontitis. Even the *aggressive* forms of periodontitis are chronic conditions, *not* acute.
- *Whether the condition is localised or generalised.* The 30% rule is a guide, but is somewhat arbitrary, so common sense in the application of this term is recommended.
- *The severity of the condition.* There

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轻度、中度或重度的定义有许多提议，但都没有严格且可靠的尺度标准。使用一个定义系统是有用的，但是像"轻度"、"中度"和"重度"这些描述性词语，虽然在目的上是有用的，但对于不同的临床医师可能指的是完全不同的情况。用以帮助描述疾病严重程度的两个测量值是临床测量的附着丧失和X线片证实的骨丧失。以下是怎样描述疾病严重程度的建议。

– 轻度：1～2 mm的附着丧失或骨丧失达根长的1/3。

– 中度：3～4 mm的附着丧失或骨丧失达根长的1/2。

– 重度：5 mm以上的附着丧失或骨丧失大于根长的1/2和（或）有根分叉病变。

有的系统用牙周袋深度作为定义疾病严重程度的方法，但是像早先论述的一样，在采用这种测量法时存在一个固有的问题，它无法区别真性和假性牙周袋，从而可能过低或过高评估疾病。

are no hard and fast rules here either and there are many suggestions about defining whether disease is mild, moderate or severe. Using a system is useful, however, as the descriptive words "mild", "moderate" and "severe", though intentionally useful, may mean entirely different things to different clinicians. Two measurements used to aid description of the severity are the clinical measure of LOA and radiographic evidence of *bone loss*, and the following is a suggestion of how severity may be described.

- *mild disease*: LOA of 1-2mm or loss of the bone covering up to one-third of the root length.
- *moderate disease*: LOA of 3-4mm or bone loss of up to one-half the root length
- *severe disease*: LOA of 5+mm or bone loss greater than one-half of the root and/or inclusion of furcation involvement.

Some systems use pocket depth as a means of defining disease severity, but as discussed earlier there is an in-built problem in using this measurement on its own, since it will not differentiate between true and false pockets and may thus markedly over- or underestimate the disease present.

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举一个诊断例子"广泛性中度慢性牙周炎"，尽管它可以通过补充更多的细节进一步定义，如"广泛性中度慢性牙周炎伴下颌中切牙和尖牙局限性重度感染"。这个诊断实际上在后期能被改变以反映疾病行为（它的分布或严重程度）方面的变化。

预　后

对牙周疾病进行诊断后，许多患者会顺理成章地询问他们的预后。回答时，临床医师有必要进行正当的防卫，因为预后是临床医师根据他（她）的知识和经验对可能发生的事件的"最佳猜测"。这一阶段出现的情况不确定，因为有两个未知的且极其重要的因素：

- *生物组织对治疗反应*。这在患者与患者之间有极大差别，有时，令人吃惊的是在重度疾病患者可反应良好，而明显更轻微的疾病患者却显示较差的反应。
- *患者合作*。对牙周疾病固有易感性越高时，患者的合作就显得越重要。如果患者的配合很差则治

An example of a diagnosis would be "generalised moderate chronic periodontitis", though it can be further refined by the addition of more detail, e.g. "generalised moderate chronic periodontitis with localised severe disease affecting the lower incisors and canines". This diagnosis actually means something and can be changed at a later date to reflect changes in the behaviour of the disease, its distribution or severity.

Prognosis

After diagnosis of periodontal disease many patients, perfectly reasonably, ask about their periodontal prognosis. In response, the clinician must necessarily be fairly guarded because the prognosis is basically the clinician's "best guess" about what is likely to happen based upon his/her knowledge and experience. The uncertainty of the situation arises at this stage because two vital factors are unknown:

- *The biological tissue response to treatment*. This can vary greatly from one patient to another and is sometimes surprising in that severe conditions may respond well, while apparently more trivial conditions show a poor response.
- *Patient co-operation*. The higher the innate susceptibility to periodontal disease the more important becomes the

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疗将不会成功，患者和医师都必须认识这一事实。不管专业护理有多好，都不能补偿较差的家庭日常护理。

patient's co-operation. If this is poor, treatment will not succeed and this fact must be recognised by both the patient and the clinician. No matter how good the professional care may be, it cannot compensate for poor home care.

但是通过对这些确定的一般预后因素和局部预后因素的思考，给出预后也是可能的。

It is possible to give a prognosis, however, by consideration of certain general and local prognostic factors.

一般预后因素

General Prognostic Factors

1. *患者的全身健康和免疫状况。* 由于许多系统性疾病对牙周膜有负面影响，所以患者的全身健康和免疫状况很重要。系统性疾病包括多种血液病、胃肠道疾病和内分泌疾病。这些疾病的发生可降低预后。为控制系统性疾病，许多患者服用的药物也可对牙周膜产生有害作用，例如细胞毒性药物和免疫抑制药物，这类药物如苯妥英有引起牙龈增生的副作用（见第 4 章）。

1. *The systemic health and immune status of the patient.* This is important, as it is known that many systemic conditions have negative consequences for the periodontium. Examples include many haematological, gastrointestinal and endocrine diseases. Presence of such conditions may reduce the prognosis. Drugs taken by many patients to control their systemic disease may also adversely affect the periodontium. Examples include cytotoxic and immunosuppressive medication and certain drugs such as phenytoin, which may have the undesired side effect of causing gingival overgrowth (see Chapter 4).

2. *遗传。* 在许多形式的牙周疾病中评价遗传学的作用是困难的，因为牙周疾病是多基因的疾病，伴有许多影响潜在的遗传易感性的混乱的环境因素（如吸

2. *Heredity.* The role of genetics in many forms of periodontal disease is difficult to evaluate because periodontal diseases are polygenic, with many confounding environmental factors (e.g.

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烟）。有证据表明在几种牙周炎类型，特别是在部分侵袭性类型中，遗传起着重要作用。询问患者是否有牙周疾病的家族史是有价值的，尤其是患者发生侵袭性／重度牙周疾病时。

smoking) influencing underlying genetic susceptibility. There is evidence of a significant role for genetic transmission in several types of periodontitis, in particular in some of the aggressive forms. It is always worth asking patients whether there is a family history of periodontal disease and this is especially worthwhile if the patient has presented with aggressive/severe disease.

3. *牙周炎的病因和类型*。很明显，牙周疾病的侵袭性越强预后越差。

3. *The aetiology and form of periodontitis*. Clearly the more aggressive the type of periodontal disease present the lower the prognosis.

4. *有附着丧失的患者的年龄*。这包括易感性的概念。如果患者是年轻人，菌斑控制良好但却有重度牙周疾病，很明显其预后比具有相同水平的牙周破坏，菌斑控制不良的老年患者要差得多。由于疾病易感性通过治疗不能改变，所以临床工作者应特别关注每个患者对牙周疾病固有的易感性。总之，易感性越高，预后将越差，治疗的侵入性也不得不越强，疾病控制后复诊时间间隔就越短。

4. *The age of the patient in relation to the attachment loss*. This encompasses the concept of susceptibility. If the patient is young and has good plaque control yet has severe disease it is clear that the prognosis is much poorer than if a similar level of destruction was seen in an elderly patient with poor plaque control. The clinician should specifically consider the innate susceptibility of *each* patient to periodontal disease, as this cannot be changed by treatment. In general, the higher the susceptibility the lower the prognosis will be, the more aggressive the treatment is likely to have to be and the shorter recall times must be if and when the disease is controlled.

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5. *患者是否吸烟*。现在有越来越多的令人信服的证据表明吸烟对牙周膜有高度的破坏作用（见第 4 章、第 7 章）。吸烟无疑降低了牙周病的预后。

6. *患者的合作态度*。毫无疑问，成功的牙周治疗依赖于患者的合作，这对高牙周病易感性患者更为重要，不良依从性将降低易感病例的预后。

7. *治疗方式的有效性*。有许多新的或附属的牙周治疗的特殊治疗方式，在特定的环境应用一些治疗可积极地影响预后。（进一步的论述请看《为牙周组制定治疗计划》和《炎症性牙周疾病的非手术治疗》）。

8. *临床医师的知识和经验*。这不意味专家应该提供所有的牙周护理，但是如果发现疾病是严重而复杂的，应该考虑将患者转诊给有专科知识和技术的医师。

5. *Whether the patient is a smoker*. There is now mounting and convincing evidence for the highly destructive effect of smoking on the periodontium (see Chapters 4 and 7). Smoking certainly reduces the periodontal prognosis.

6. *The attitude and co-operation of the patient*. There is no doubt that a successful outcome from periodontal treatment is dependent upon patient cooperation and that this becomes more important in patients with a high innate susceptibility to periodontal disease. Poor compliance will reduce prognosis in susceptible cases.

7. *The availability of treatment modalities*. There are many new forms of, or adjuncts to, periodontal therapy and the application of some in particular circumstances may positively affect the prognosis. (For further discussion please see *Quintessentials: Treatment Planning for the Periodontal Team* and *Non-Surgical Management of Inflammatory Periodontal Diseases*.)

8. *The knowledge and experience of the clinician*. This does not mean that specialists should provide all periodontal care, but if the disease found is severe or complicated in any way, consideration should be given to referral of the patient to someone with specialist knowledge and skills.

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9. *制定一个适当的维持治疗计划。* 牙周疾病不能治愈但能稳定在最佳状态。因为患者固有的疾病易感性在治疗后并未改变，即使疾病得到控制，在某些病例疾病也可能将再次发生。为了早期发现任何这类复发并使治疗需求保持简单，需要制定一个好的维持计划。当一个灵活的、令人满意的和个别设计的维持计划不能建立时将肯定降低长期预后。

9. *Institution of an adequate maintenance programme*. Periodontal disease cannot be cured and can at best be stabilised. As the innate susceptibility of a patient to their disease remains unchanged after treatment, even if the disease is controlled, it is possible – and in some cases likely – that the disease will recur. A good maintenance programme is required so that any such recurrence can be detected early while the treatment needs remain simple. Failure to institute a flexible, competent and individually designed maintenance plan will certainly reduce the long-term prognosis.

局部预后因素

1. *菌斑量。* 如果菌斑水平在患者个体疾病阈值下，说明在个体口内菌斑控制是充分的，患者疾病得到控制。充分控制因而也是非常个体化的概念，它直接将他或她自己的菌斑与个体疾病易感性相联系，如果菌斑控制不充分，则预后降低。这个概念支持非特异性菌斑假说。

2. *菌斑中的微生物。* 有令人信服的证据表明某些菌斑细菌如伴放线放线杆菌是非常致命的

Local Prognostic Factors

1. *The amount of plaque present*. The plaque control in an individual mouth is adequate if the plaque level lies below the individual disease threshold for that patient and disease is controlled. Adequate control is thus a very individual concept and relates directly to the susceptibility of the individual to his or her own plaque. If the plaque control is inadequate the prognosis is reduced. This concept supports the *Non-Specific Plaque Hypothesis*.

2. *The organisms present in plaque*. There is compelling evidence that certain plaque bacteria such as *Actino-*

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（见第2章）。这些微生物被描述为具有“一个低的临界感染”，指只需相对很少的量就能致病。它们的存在会降低预后，这个概念支持特异性菌斑学说。

bacillus actinomycetemcomitans are particularly virulent (see Chapter 2). These organisms are described as having "a low critical infection", meaning that relatively few are required to cause a problem. Their presence reduces the prognosis and this concept supports the *Specific Plaque Hypothesis*.

3. *牙周袋深度和位置*。牙周袋越深治疗越困难，预后越差。牙周袋的位置也有关系，因为牙周袋在口内的位置越靠后越不容易接近。由于前牙区段的入路是最少被限制的，因此这些位点的预后也普遍较好。

3. *Pocket depth and location*. The deeper the pocket the more difficult it is to treat and the poorer the prognosis. The location of the pocket is also relevant as the ease of access reduces further back in the mouth. The access is thus least restricted in the anterior segments and the prognosis is generally improved in these sites.

4. *附着丧失的数量和分布*。很明显这些与预后呈负相关关系。

4. *Amount and expanse/distribution of loss of attachment*. These are clearly negatively related to prognosis.

5. *根分叉病变的发生和严重程度*。这与预后呈负相关关系（见第8章）。

5. *Presence and severity of furcation involvement*. This is negatively related to prognosis (see Chapter 8).

6. *修复情况*。不良修复体边缘、不良设计的义齿和正畸矫正器都将降低预后，因为它们使充分菌斑控制难以获得。

6. *The restorative condition*. The presence of poor restoration margins, poorly designed dentures and orthodontic appliances will all tend to reduce the prognosis as they make achieving adequate plaque control difficult if not virtually impossible.

7. *牙髓状况*。如果一颗牙有牙髓病变和牙周病变，它的预后会

7. *The endodontic condition*. If a tooth has both endodontic and periodontal

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降低，因为在开始牙周治疗前需要拔除患牙或对患牙进行完善的根管治疗。许多因素包括牙齿在口内的位置、牙根弯曲度、根管阻塞等降低了成功完成RCT的机会。（进一步的论述见《牙髓根管感染的治疗》）。

involvement its prognosis is reduced, as it will need to be either extracted or have successful root canal therapy (RCT) performed before the periodontal work can begin. The chance of successful RCT is reduced in its own right by many factors including location within the mouth, root curvature, presence of obstructions in the canal, etc. (For further discussion see *Quintessentials: Managing Pulp Canal Infection*).

8. *牙周疾病的活跃性*。活跃的位点越多预后越差。

8. *The activity of the periodontal disease*. The more active sites present the lower the prognosis.

9. *牙根长度*。如果牙根短，骨丧失或附着丧失的毫米数比牙根长的情况相对来说要严重得多。所以短根牙齿比较长牙根的类似牙的预后差。

9. *The root length*. If the roots are short, a given number of millimetres of bone loss or loss of attachment is relatively much more serious than if the roots are long. Short-rooted teeth thus tend to have a lower prognosis than similar teeth with longer roots.

10. *牙根形状*。牙根凹槽和沟促进菌斑积聚并降低易感患者的预后。

10. *The root shape*. The presence of root grooves and furrows promotes plaque accumulation and reduces prognosis in susceptible patients.

11. *牙齿位置异常*。牙齿拥挤和倾斜能增加菌斑聚集，降低菌斑控制措施的效果，导致易感患者的预后降低。

11. *Anomalies of tooth position*. Crowding or tilting of teeth can increase plaque accumulation and reduce the effectiveness of plaque control measures resulting in reduced prognosis in susceptible patients.

12. *牙石*。尽管牙石是不活动的，

12. *Presence of calculus*. Although calcu-

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但它具有粗糙的表面，能够促进菌斑聚集，对易感患者将降低预后。

lus is inert, it has a rough surface, which promotes plaque accumulation, which in a susceptible patient will reduce prognosis.

经过对上述所有问题的思考将使临床医师对准确的牙周诊断和预后问题作出合理的回答。

Consideration of all the above will allow the clinician to give a reasoned response to the issues of accurate periodontal diagnosis and prognosis.

拓展阅读

Further Rdeding

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